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July
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VOLUME XXXVII • NUMBER 7

Heme Pigment Metabolism
J. Chandler Smith, M.D.

Treatment of Vaginitis
J. C. Walsh, M.D., J. B. Sheffery, M.D., and T. A. Wilson, M.D.

Automatic Spirographic Analysis
W. R. Ayers, M.D., C. Ries, M.D., and C. A. Caceres, M.D.

Emergency Room and Health Needs of Children
N. Estampador, M.D., A. D. Kessler, M.D., and R. B. Scott, M.D.

Metastatic Carcinoma of Ciliary Body
P. I. Matias, M.D., and B. I. Shnider, M.D.

Medical Expert and Printed Page
Maurice H. Herzmark, M.D.

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Annual Scientific Assembly

WASHINGTON HILTON HOTEL
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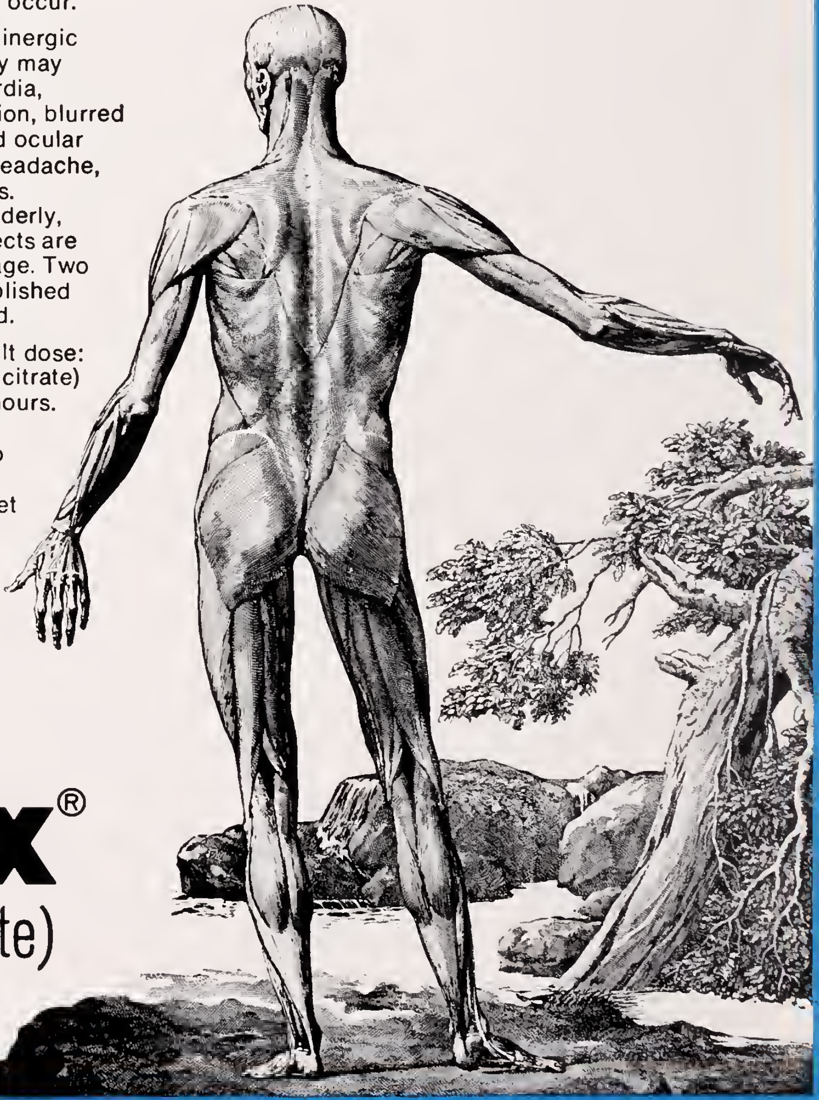

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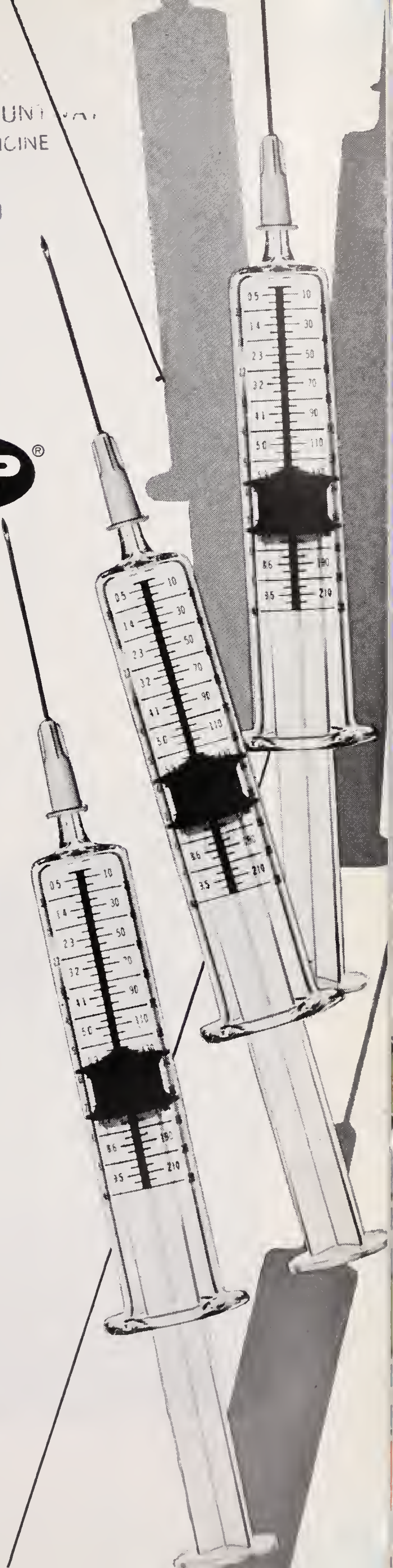

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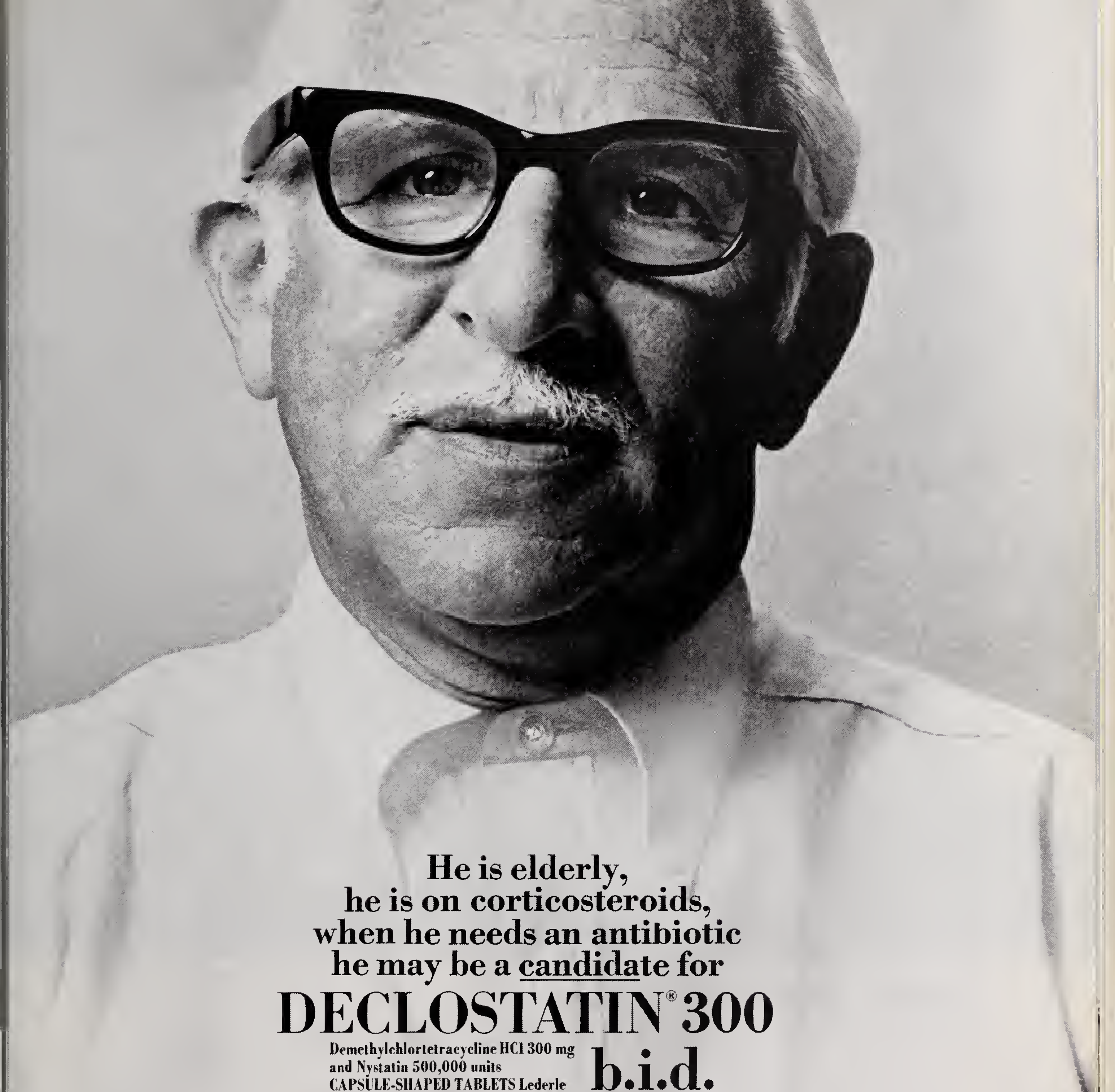

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rning: In renal impairment, usual doses may lead to excessive accumu-
on and liver toxicity. Under such conditions, lower than usual doses
indicated, and, if therapy is prolonged, serum level determinations
y be advisable. A photodynamic reaction to natural or artificial sun-
it has been observed. Small amounts of drug and short exposure may
duce an exaggerated sunburn reaction which may range from ery-
ma to severe skin manifestations. In a smaller proportion, photo-
rgic reactions have been reported. Patients should avoid direct
osure to sunlight and discontinue drug at the first evidence of skin
comfort. Necessary subsequent courses of treatment with tetracy-
es should be carefully observed.

cautions: Overgrowth of nonsusceptible organisms may occur. Constant observation is essential. If new infections appear, appropriate measures should be taken.

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Side Effects: Gastrointestinal system—anorexia, nausea, vomiting, diarrhea, stomatitis, glossitis, enterocolitis, pruritus ani. Skin—maculopapular and erythematous rashes; a rare case of exfoliative dermatitis has been reported. Photosensitivity; onycholysis and discoloration of the nails (rare). Kidney—rise in BUN, apparently dose related. Transient increase in urinary output, sometimes accompanied by thirst (rare). Hypersensitivity reactions—urticaria, angioneurotic edema, anaphylaxis. Teeth—dental staining (yellow-brown) in children of mothers given this drug during the latter half of pregnancy, and in children given the drug during the neonatal period, infancy and early childhood. Enamel hypoplasia has been seen in a few children. If adverse reaction or idiosyncrasy occurs, discontinue medication and institute appropriate therapy. Average Adult Daily Dosage: 150 mg q.i.d. or 300 mg b.i.d. Should be given 1 hour before or 2 hours after meals, since absorption is impaired by the concomitant administration of high calcium content drugs, foods and some dairy products. Treatment of streptococcal infections should continue for 10 days, even though symptoms have subsided.

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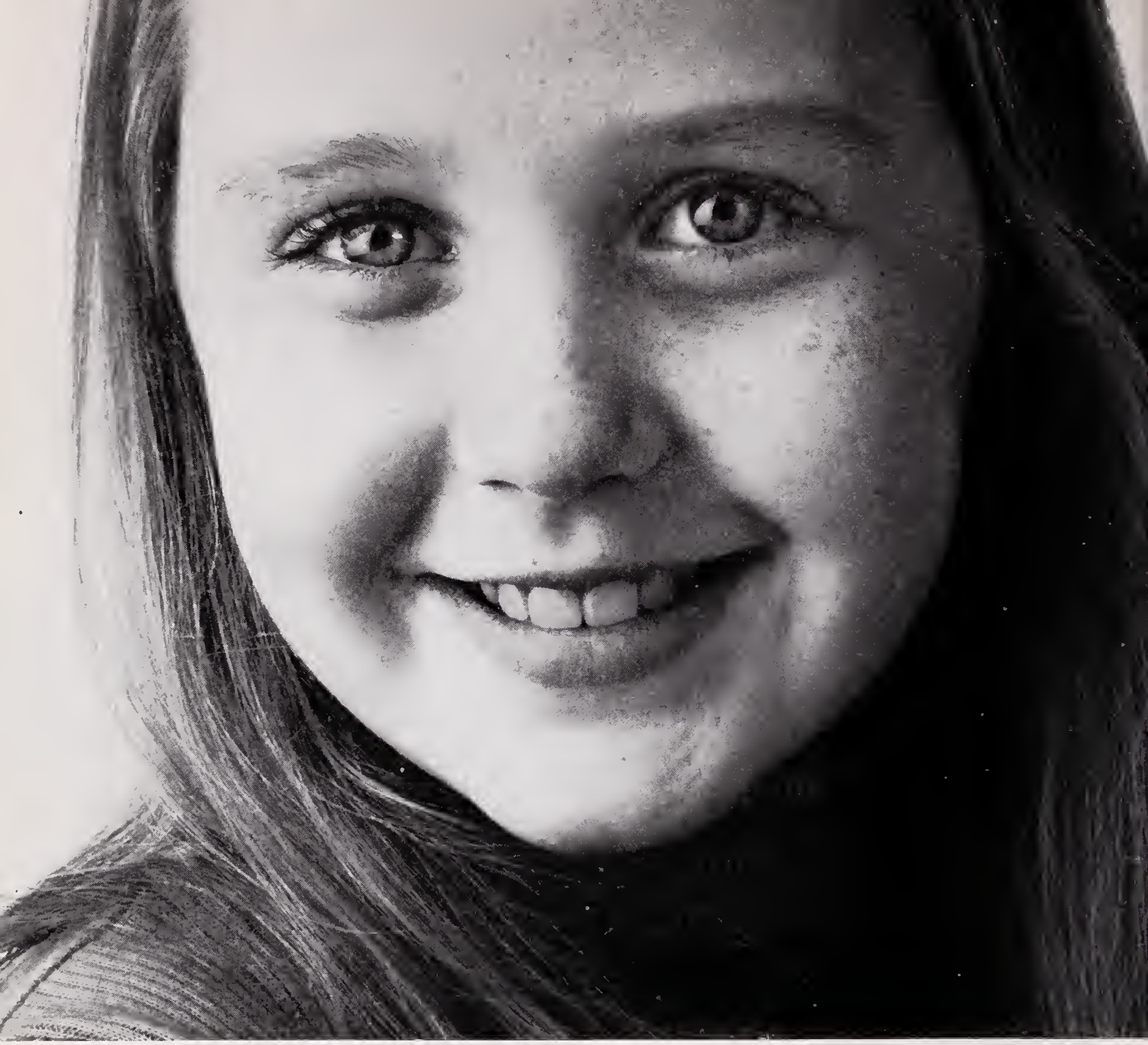

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Warnings: Acute anaphylaxis (may prove fatal unless promptly controlled) is rare but more frequent in patients with previous penicillin sensitivity, bronchial asthma or other allergies. Resuscitative (epinephrine, aminophylline, pressor amines) and supportive (antihistamines, methylprednisolone sodium succinate) drugs should be readily available. Other rare hypersensitivity reactions include nephropathy, hemolytic anemia, leucopenia and thrombocytopenia. In suspected hypersensitivity, evaluation of renal and hematopoietic systems is recommended.
Precautions: In suspected staphylococcal infections, perform proper laboratory studies including sensitivity tests. If overgrowth of nonsusceptible organisms occurs (constant observation is essential), discontinue penicillin and take appropriate measures. Whenever allergic reactions occur, withdraw penicillin unless condition being treated is considered life threatening and amenable only to penicillin. Penicillin may delay or prevent appearance of primary syphilitic lesions. Gonorrhea patients suspected of concurrent syphilis should be tested serologically for at least 3 months. When lesions of primary syphilis are suspected, dark-field examination should precede use of penicillin. Treat beta-hemolytic streptococcal infections with full therapeutic dosage for at least 10 days to prevent rheumatic fever or glomerulonephritis. In staphylococcal infections, perform surgery as indicated.
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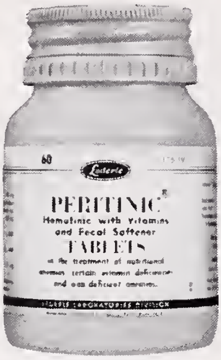

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August 11–15: National Medical Association. Shamrock-Hilton, Houston. Address: Mr. Samuel C. Smith, Administrative Secretary, 520 W Street, N.W., Washington, D. C. 20001.

August 25–31: International Congress of Physiological Sciences. Washington. Address: Secretariat, International Congress of Physiological Sciences, 9650 Rockville Pike, Bethesda, Md. 20014.

September 1–6: American Society of Hematology. Waldorf-Astoria, New York. Address: Dr. J. F. Mustard, Department of Pathology, McMaster University, Hamilton, Ontario.

September 5–7: American Association of Obstetricians and Gynecologists. The Homestead, Hot Springs, Va. Address: Dr. Robert B. Wilson, Executive Secretary, 200 First Street, S.W., Rochester, Minn. 55901.

September 6–7: Cardiovascular Symposium sponsored by Tidewater Heart Association and Council on Clinical Cardiology, American Heart Association. Lake Wright Motel, Virginia Beach, Va. Address: Dr. James D. Price, Chairman, 523 Boush Street, Norfolk, Va. 23510.

September 13–20: American Academy of General Practice. Las Vegas, Nevada. Address: Mr. Mac F. Cahal, Executive Director, Volker Blvd. at Brookside, Kansas City, Mo. 64112.

September 15–20: International Congress on Alcohol and Alcoholism. Shoreham Hotel, Washington. Address: Secretariat Office, 1130 17th Street, N.W., Washington, D. C. 20036.

September 19–22: American Medical Writers' Association. Mayflower Hotel, Washington. Address: Mr. Stephen T. Donohue, Treasurer, 420 Lexington Avenue, New York, N. Y. 10017.

October 4–8: International Congress on Diseases of the Chest. Washington Hilton Hotel, Washington. Address: American College of Chest Physicians, 112 East Chestnut Street, Chicago, Ill. 60611.

October 14–18: American College of Surgeons. Dennis Hotel, Atlantic City, N. J. Address: Dr. John Paul North, 55 East Erie Street, Chicago, Ill. 60611.

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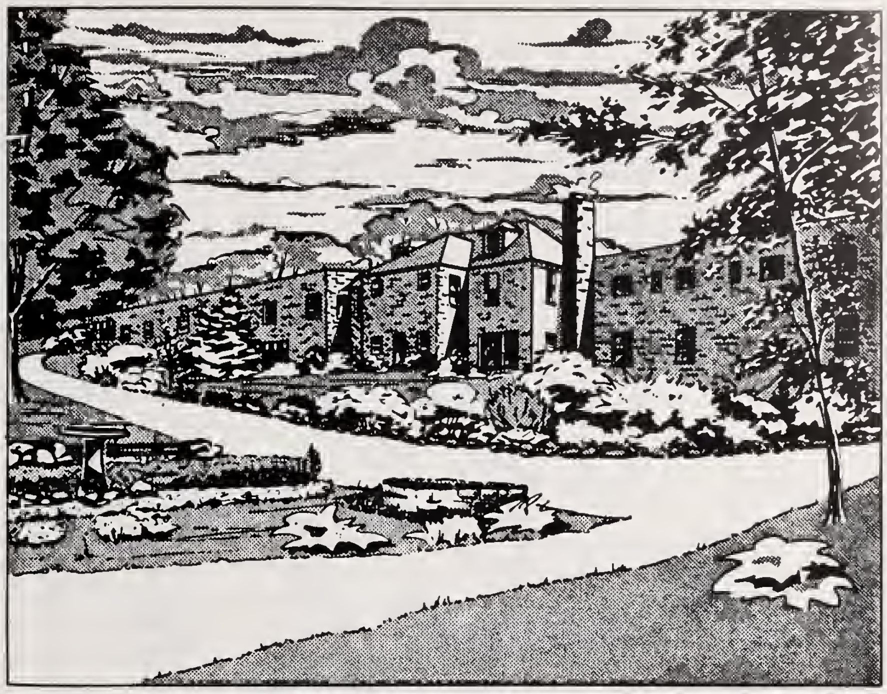

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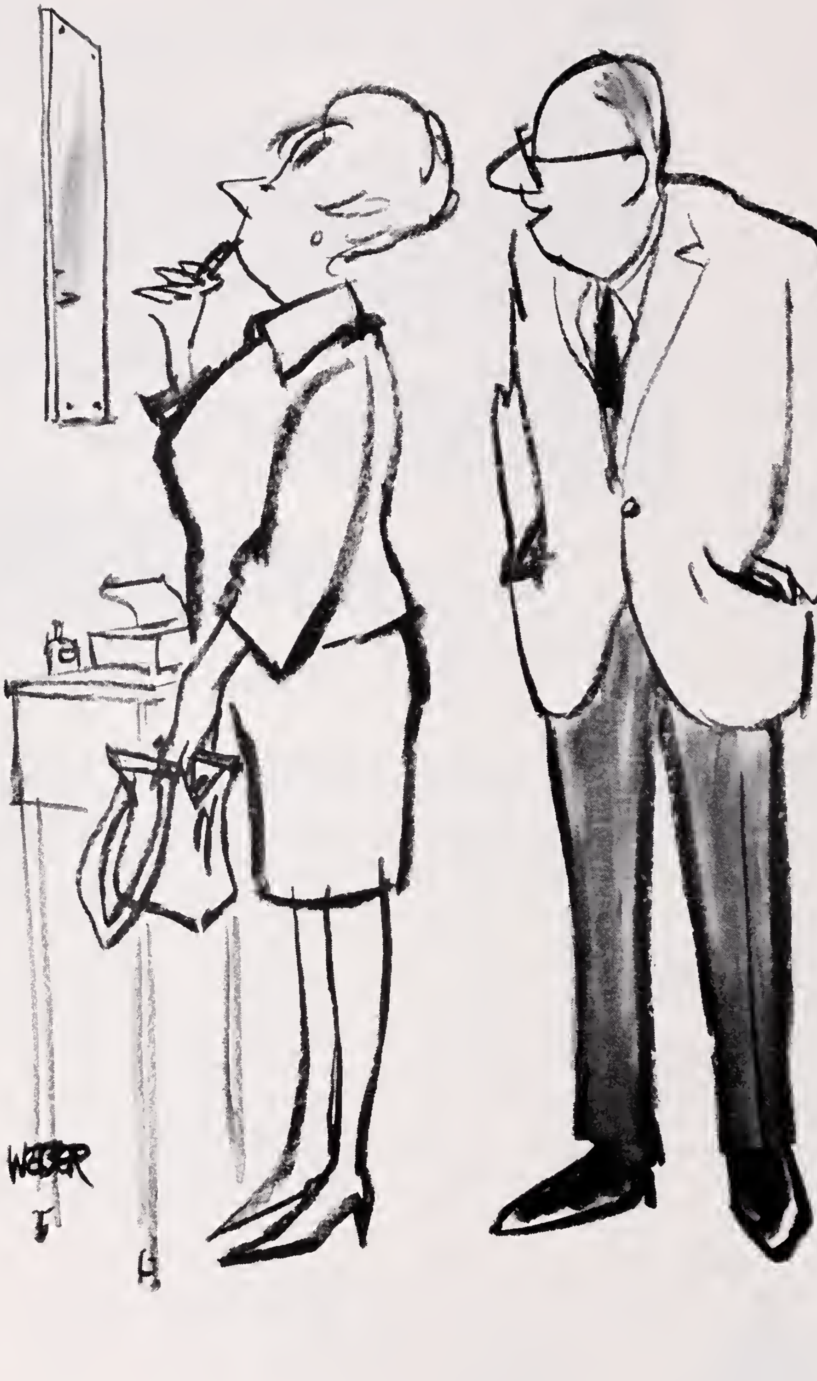

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Side Effects: Overstimulation of the central nervous system, jitteriness and insomnia or drowsiness. Dextro-amphetamine sulfate: Insomnia, excitability, and increased motor activity are common and ordinarily mild side effects. Confusion, anxiety, aggressiveness, increased libido, and hallucinations have also been observed, especially in mentally ill patients. Rebound fatigue and depression may follow central stimulation. Other effects may include dry mouth, anorexia, nausea, vomiting, diarrhea, and increased cardiovascular reactivity.

Meprobamate: Drowsiness may occur and can be associated with ataxia; the symptom can usually be controlled by decreasing the dose, or by concomitant administration of central stimulants. Allergic or idiosyncratic reactions: maculopapular rash, acute nonthrombocytopenic purpura with petechiae, ecchymoses, peripheral edema and fever, transient leukopenia. A case of fatal bullous dermatitis, following administration of meprobamate and prednisolone, has been reported. Hypersensitivity has produced fever, fainting spells, angioneurotic edema, bronchial spasms, hypotensive crises (1 fatal case), anuria, stomatitis, proctitis (1 case), anaphylaxis, agranulocytosis and thrombocytopenic purpura, and a fatal instance of aplastic anemia, but only when other drugs known to elicit these conditions were given concomitantly. Fast EEG activity, usually after excessive dosage. Impairment of visual accommodation. Massive overdosage may produce drowsiness, lethargy, stupor, ataxia, coma, shock, vasomotor and respiratory collapse.

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Warning: Clinical reports have suggested that there may be a risk of teratogenesis associated with the use of this compound during the first trimester of pregnancy. Unless, in the opinion of the prescribing physician, the potential benefits outweigh the possible risks, it should not be used during the first trimester of pregnancy. Cardiovascular complications, including myocardial infarction and arrhythmias, have occasionally occurred in susceptible individuals. Patients with cardiovascular disease should be given the drug only under careful observation and in low dosage.
Precautions: Since suicide is always a possibility in severely depressed patients and one which may persist until significant remission occurs, such patients should be carefully supervised during early treatment. Some severely depressed patients may also require hospitalization and/or concomitant electroconvulsive therapy. Because of its anticholinergic effect, caution should be observed in prescribing the drug for patients with increased intraocular pressure.
In rare instances, transient cardiac arrhythmias have occurred in hyperthyroid patients and in patients receiving thyroid medication when this compound was added to the regimen.
Imipramine may block the pharmacologic activity of guanethidine and other related adrenergic neuron-blocking agents.
The drug is not recommended at th present time in patients under 12 ye of age.
Adverse Reactions: Dryness of the mouth, tachycardia, constipation, d turbances of accommodation, swea ing, dizziness, weight gain, urinary frequency or retention, nausea and vomiting, peripheral neuritis, mild parkinson-like syndrome, tremors, rare cases of falling in elderly patients, confusional states (with suc symptoms as hallucinations and dis orientation), activation of psychosis schizophrenics and agitation (inclu ing hypomanic and manic episodes which may require dosage reductio

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and/or addition of a tranquilizer or temporary discontinuation of the drug, epileptiform seizures, orthostatic hypotension and substantial blood pressure fall in hypertensive patients, purpura, transient jaundice, bone marrow depression including agranulocytosis, sensitization and skin rash including photosensitization, eosinophilia, and mild withdrawal symptoms on sudden discontinuation after prolonged treatment with high doses. Occasional hormonal effects (impotence, decreased libido, and estrogenic effects) may be observed. Atropine-like effects may be more pronounced (e.g. paralytic ileus) in susceptible patients and in those using anticholinergic agents (including antiparkinsonism drugs).
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Availability: Round tablets of 25 and 50 mg.; triangular tablets of 10 mg. for geriatric and adolescent use; and ampuls, each containing 25 mg. in 2 cc. for I.M. administration.
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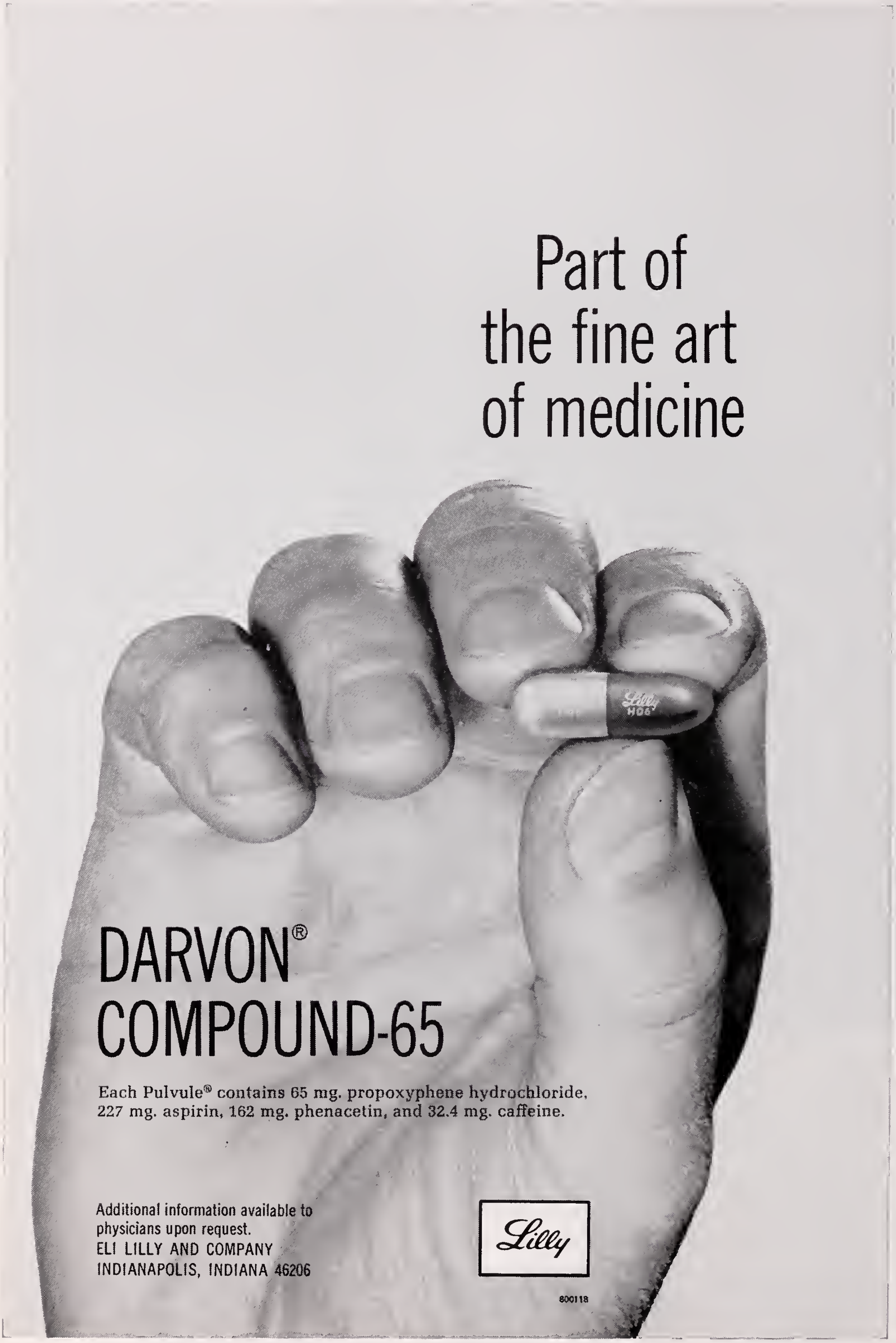

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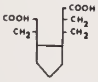
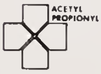
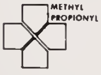
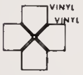
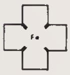
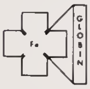
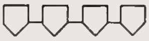
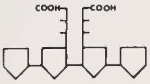
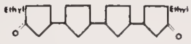
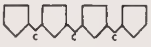
Heme Pigment Metabolism: A Clinicopathologic Spectrum

J. CHANDLER SMITH, M.D.*

ONE OF THE MOST DIFFICULT and yet intriguing of subjects for the physician is the metabolism of the heme pigments. There are so many alterations, so many clinical expressions, and so many laboratory tests that one is often confused and may fail to understand the relationship of an isolated fact to the spectrum of possibilities that the subject presents. It may therefore prove helpful to scan the spectrum, citing only the important features, so that the main points of physiology, pathology, laboratory tests, and clinical events will fall into order and a conceptual unit will emerge. In order to do this only abbreviated descriptions can be given.

TABLE 1: Synthesis and Degradation of Hemoglobin

	SUBSTANCE	STRUCTURE	IDENTIFICATION
SYNTHESIS	Porphobilinogen		Aldehyde positive, color insoluble in CCl_4
	Uroporphyrin		
	Coproporphyrin		Red flouresence in ether leyer of urine
	Protoporphyrin		
	Heme		Gueiec, H_2O_2, turns blue
	Hemoglobin		Gueiec, H_2O_2, turns blue
DEGRRADATION	Unconjugeted Bilirubin		Aldehyde reection slow, edd elcohol
	Conjugeted Bilirubin		Aldehyde reection prompt without alcohol
	Bilinogens		Aldehyde positive, color soluble in CCl_4
	Bilin		

The Physiologic Sequence

The blood pigments are formed by the assembly of a succession of substances as shown in table 1. In the beginning the amino acid glycine from the protein pool joins with succinate from the carbohydrate pool to form adipic acid. Decarboxylation of this acid yields aminolevulinic acid, 2 molecules of which combine to form the first structural entity of the porphyrin sequence as shown in table 1. That substance, the monopyrrole porphobilinogen, contains an acetate and a propionate side chain as shown. Four molecules of porphobilinogen then unite to form the basic porphyrin structure, a tetrapyrrole. Three succeeding steps now differentiate the basic porphyrin into 3 similar but different molecules. The first is the addition of acetate and propionate side chains which make uroporphyrin. As the acetate side chain is changed to the methyl radical, the molecule becomes coproporphyrin. Finally, vinyl radicals replace both of the side chains and the molecule becomes protoporphyrin, the immediate precursor of heme. Iron in the ferrous state is now locked into the central bonding to make heme, and globin is attached to the side to complete the hemoglobin molecule. These events take place largely in the bone marrow in the maturing cells of the erythrocytic series. Thus the immature cell contains more protoporphyrin than hemoglobin, while in the mature erythrocyte

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these proportions are reversed. A normal adult synthesizes about 8 grams of hemoglobin daily.

About 120 days after synthesis the red blood cell breaks down in the reticuloendothelial cells of the liver, spleen, and bone marrow, iron is extracted from the molecule, globin is detached, and both are released for reutilization. The remaining porphyrin, however, is destined for degradation and excretion. The first step is a splitting open of the cyclic structure so that a linear tetrapyrrole configuration is left. This substance, bilirubin, is now discharged from the reticuloendothelial cells to enter the blood stream where it attaches to the albumin component of the plasma proteins. Regardless of this attachment, it is considered as "free" bilirubin; it has not yet reached the liver which is its next destination. The "free" or pre-liver bilirubin is insoluble in water. On arrival at the liver, the hepatocytes detach the albumin, conjugate the tetrapyrrole with glucuronic acid, break it into 2 dipyrrole structures, and transfer the new substance into the bile canaliculi. Without albumin, the post-liver or conjugated bilirubin is soluble in water. The conjugated bilirubin passes into the gallbladder for concentration and into the gut for further change. In the small intestine, bacteria reduce the bile to a wide variety of bilinogens which are collectively known as urobilinogen. Most of these are absorbed into the enterohepatic circulation; the liver cells again extract them from the blood, and in the transfer through the hepatic cells the urobilinogen is reconstituted as bilirubin and again excreted into the bile channels. Some of the absorbed urobilinogen, however, passes by the columns of hepatic cells and through the liver to enter the systemic circulation. This portion is excreted by the kidney, the urine normally containing 0 to 4 mg. per 24 hours. The urobilinogen that is not absorbed in the ileum is oxidized to the bilins (urobilin and stercobilin) which are excreted and account for the brown color of the feces.

Thus the sequence is completed. If 8 Gm. of hemoglobin is synthesized each day, 8 Gm. of hemoglobin is also broken down each day. Since the porphyrin component represents 3.5 per cent of the hemoglobin molecule, 0.28 Gm. or 280 mg. of bilirubin is formed daily. Only 0 to 4 mg. appears in the urine; the remainder passes out in the feces in the usual amount of 100 to 250 mg. per day.

This account purposely omits much information, arbitrarily selecting those facts that appear most useful to the clinical and laboratory diagnosis of disorders of porphyrin-pigment metabolism. Detailed accounts are given by Cantarow and Trumper[1] and by Cantarow and Shepartz.[2]

The Laboratory Procedures

Certain laboratory tests permit the recognition and quantitation of various members of this metabolic chain. Of these, 6, as indicated in table 1, are especially useful. Although the techniques of these tests are thoroughly established so that no detailed account need be given here, certain comments are in order. The first is that Ehrlich's aldehyde reagent is useful in identifying 2 different members located at opposite ends of this metabolic chain. On addition of the aldehyde reagent to urine a red color develops with both porphobilinogen and urobilinogen. The distinctive feature is that the former is insoluble in chloroform and the latter is not. With the addition of chloroform to the tube, therefore, the red color remains in the upper aqueous phase with porphobilinogen and sinks to the lower nonaqueous phase with urobilinogen. Specific tests are available for uroporphyrin and for coproporphyrin.[3] Once the iron molecule is incorporated into the developing structure the guaiac test becomes positive. This is because the addition of iron forms heme, heme includes peroxidases, and in the presence of an oxygen source (hydrogen peroxide) the guaiacetic acid is oxidized and turns blue.[4] Therefore, heme and hemoglobin are both associated with a positive guaiac test.

The third comment pertains to pre-liver and post-liver bilirubin. The former has not yet reached the liver and the pigment is accordingly still attached to albumin. It is therefore insoluble in water. Because of this insolubility the addition of Ehrlich's aldehyde reagent produces a red color that develops slowly but is hastened by the addition of alcohol (indirect reacting). It is in the transit through the hepatic cells that the bilirubin is separated from its albumin, conjugated with glucuronic acid, and split into 2 dipyrroles. After these reactions have taken place the pigment is transferred into the bile ductules. It may, nevertheless, accumulate in the blood. Without albumin the conjugated pigment is soluble in water so that the development of a red color on the addition of the aldehyde reagent is prompt without the addition of alcohol (direct reacting). Thus a single test indicates the proportion of circulating pigment that has accumulated before, or has passed through, the interposed liver. It is, of course, not to be overlooked that only post-liver conjugated bilirubin, because of its water insolubility, may appear in the urine.

The Pathologic Processes

The pathologic alterations of the blood pigments may be visualized in 2 broad phases, the former representing the abnormalities of formation and extending to the fully constructed hemoglobin molecule, and the latter representing the abnormalities of breakdown and including those substances that the degradative process furnishes. A horizontal line thus separates the metabolic chain into 2 broad categories. The former are the synthesizing abnormalities, the latter the degradative abnormalities. The former consists of the porphyrin syndromes, the latter the illnesses accompanied by jaundice.

The synthesizing abnormalities. Natural porphyria is due to an inborn error in the synthesis of heme in which the bone marrow (p. erythropoietica) or the liver (p. hepatica) produces an excess of uroporphyrin. With the hepatic type of porphyria the tetrapyrrole uroporphyrin splits apart in the urine to form the monopyrrole, porphobilinogen. Therefore, although the excess metabolite in the blood and urine is principally uroporphyrin, the substance readily identified in the urine is porphobilinogen. In contrast to this inborn metabolic disturbance, porphyria may be acquired. It is a minor accompaniment of those conditions associated with increased red blood cell production. This is not altogether unexpected as even mature erythrocytes contain some protoporphyrin and traces of coproporphyrin. The important acquired porphyrinuria, however, is induced by the ingestion of lead. Lead inhibits the synthesis of heme, coproporphyrin accumulates, and large amounts are excreted in the urine. While lead may be specifically quantitated in the urine, coproporphyrin may be identified by a red fluorescence in the ether layer of urine that has been acidified and shaken. The method is a convenient and widely used screening test for plumbism.[5]

No account is given here of the synthesizing abnormalities that affect the globin portion of the hemoglobin molecule and cause such conditions as sickle-cell anemia and thalassemia.

The degradative abnormalities. Although bilirubin is formed by the reticuloendothelial cells scattered throughout the body, and although degradation of heme pigment involves several steps and alternate pathways, the narrow channel through which all bilirubin must pass is the liver. The bilirubin in the serum enters the liver through the portal vein and hepatic artery; within the liver these channels fuse so that all blood passes down the sinusoids of the hepatic lobule. The flow of bile may be thought of as taking place in the opposite direction within a canaliculus on the other side of the cord of hepatic cells. Thus the hepatic cell sits between the blood and bile, and a principal function of this cell is to extract bilirubin from the former, conjugate it with glucuronic acid and transfer it to the latter, whence it will be excreted from the liver. Figure 1 presents a diagram of this transition.

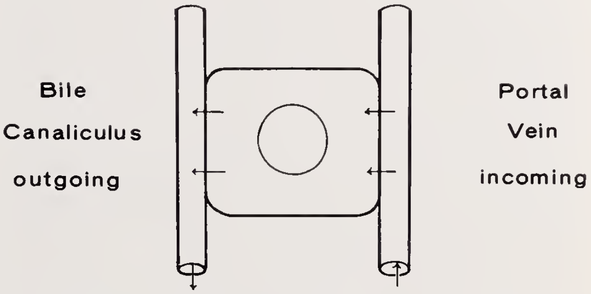

FIG. 1. The blood-bile hepatic transition.

Disturbances of heme degradation may be considered according to the position of the hepatic cell between the blood and bile, one surface being in contact with each. Increase of bilirubin in the blood may thus result from overproduction, failure to enter the hepatic cell, failure to conjugate within the hepatic cell, failure to leave the hepatic cell, and reëntry of the pigment into the blood after passing through the hepatocyte because of liver-cell injury or biliary obstruction. All are thus characterized by jaundice. These mechanisms and the syndromes they represent are shown in table 2.

TABLE 2: Degradative Abnormalities

Mechanism	Condition
1. Overproduction	Hemolytic processes
2. Failure to enter	Gilbert's disease
3. Failure to conjugate	Neonatal jaundice (Crigler-Najjar syndrome)
4. Failure to leave	Dubin-Johnson syndrome (chronic idiopathic jaundice)
5. Reëntry	Hepatic-cell disease (biliary obstruction)

Hemolytic processes that produce excessive bile pigment include the hemolytic anemias, hemolytic transfusion reactions, and absorption of large collections of extravasated blood as in pulmonary infarcts and hematomas. The cause of Gilbert's disease or mild constitutional hepatic dysfunction is not known although it is suggested that failure of uptake by the hepatic cell may be at fault. The jaundice of premature and newborn infants is likely due to an insufficiency of glucuronyl transferase since the enzyme systems of fetal and neonatal livers do not approach adult levels of activity until about 10 days after birth.[2] The Crigler-Najjar syndrome is congenital, usually fatal, and occurs in children. The Dubin-Johnson and chronic idiopathic jaundice syndromes are both rare and are both attended by a slight increase of conjugated pigment in the blood. The former is accompanied by an unknown pigment that accumulates in the hepatic cells; the latter is familial and without hepatic-cell pigmentation. Reëntry jaundice may be due to hepatic-cell injury with a break in the blood-bile barrier or to increased hydrostatic pressure within the bile capillaries that drives the pigment back through the hepatic cells into the blood. Although reëntry jaundice is easily the most common, the precise mechanism of its formation is not known.

The Clinical Diagnosis

The preceding information may now be assembled so that clinical diagnosis is facilitated. With erythropoictic porphyria the patient is an infant, the urine is red, the skin is blistered from photosensitivity, and the teeth are stained brownish-red from the deposition of heme precursor. The condition is extremely rare, only 42 cases being cited by Harris.[5] In the hepatic type of porphyria the patient is an adolescent or adult, the illness is intermittent, and the symptoms are of abrupt onset with abdominal pain and motor and sensory nerve involvement predominating. The urine turns dark red on standing, contains porphobilinogen, and gives a positive aldehyde reaction with the insoluble pigment remaining in the upper aqueous layer. The course is irregular with many remissions and exacerbations.

Acquired porphyria accompanies those conditions associated with rapid increase of red blood cell production such as infections, malignant tumors, hepatitis, and cirrhosis. In all of these coproporphyrin is slightly increased in the serum. The important acquired disturbance of porphyrin metabolism, however, is that due to lead poisoning. The clinical picture is characteristic—acute abdominal colic, neurologic disorders including foot- or wrist-drop, encephalopathy, and lead line on the gums associated with a pronounced normocytic anemia. Aside from inhibiting heme synthesis, the lead is inert but mobile. It is metabolized like calcium and is deposited in bone where an epiphyseal lead line may be seen on radiographic examination. It clings tenaciously to erythrocytes so that excretion is extremely slow and years are required for complete elimination. Specific diagnosis requires the identification of lead in the urine although the presence of coproporphyrin is sufficient for a working diagnosis.[5] At autopsy examination the pathognomonic observation is the presence of inclusions in the nuclei of the hepatic and renal tubular cells.

The degradative syndromes are characterized by jaundice. The first is due to an overproduction of bilirubin as indicated in table 2. Distinct disease processes are associated with the icterus, and especially common are massive bleeding, pulmonary infarction, and hemolytic anemia. The pigment is "free" and "pre-liver" so that it is insoluble in water and cannot be excreted by the kidney. Therefore, bilirubin is absent from the urine (acholuric), and this is the outstanding clinical feature. The excess pigment is routed into the bowel so that the daily excretion of urobilinogen in the stool is markedly increased. The liver is normal, however, and the pigment absorbed from the gut that is returned to the liver in the enterohepatic circulation is redirected into the biliary channels so that the urobilinogen excreted in the urine is only slightly increased. Thus, the conspicuous clinical features are jaundice, a disease process associated with hemoglobin breakdown, and the absence of bilirubin in the urine.

An uncommon cause of slight jaundice in adults is known as Gilbert's disease or constitutional hepatic dysfunction. The icterus is slight, the patient is otherwise well, and the excretion of urobilinogen in urine and stool is normal. Moreover, the structure of the liver and the rate of erythrocyte destruction are normal. The condition is attributed to inability of the bilirubin to enter the hepatic cells.[4]

Once the pigment has entered the liver cell, conjugation with glucuronic acid may be faulty. This occurs in the newborn during the first few days of life before the enzyme necessary for conjugation is fully formed. The condition is self-limited, however, and disappears as soon as sufficient glucuronyl transferase is formed. Rarely the transferase enzyme is congenitally absent so that the pigment remains unconjugated and extremely high levels of bilirubin result. This condition, known as Crigler-Najjar syndrome, is fatal, limited to the newborn, and death is associated with pigmentation of the basal nuclei of the brain (kernicterus) comparable to that seen more commonly in erythroblastosis fetalis.

Once the bilirubin is conjugated in the hepatic cell it may still be unable to leave the cell and enter the bile ductule. Two conditions of this kind are described. In both, the bilirubin is conjugated and hence water-soluble so that it appears in the urine. In one (Dubin-Johnson syndrome) an unidentified pigment is also present in the hepatocytes. In the other (idiopathic jaundice) the liver cells appear normal and the condition has a familial distribution. Both are rare.

Most cases of jaundice are due to the reëntry of pigment into the blood after it has been conjugated and passed through the hepatic cell. Most cases of jaundice are accordingly associated with bilirubin in the urine (choluric). When the principal process is bile-duct obstruction, the conjugated pigment moves only "backward" into the blood. When the principal process is hepatic-cell injury and the bile channels are patent, the pigment moves both "backward" into the blood and "forward" into the bile ducts as well. It is in distinguishing between these 2 main pathologic processes that clinical judgment and laboratory data are especially important. In both, the pigment is conjugated and appears in the urine. In both, hepatic-cell disease may be present, but in only one is there apt to be absence of urobilinogen from the urine. In obstructive jaundice bile cannot reach the intestine, conversion to urobilinogen cannot occur, and none can be absorbed into the enterohepatic circulation to reach the liver, pass through into the systemic circulation, and be excreted in the urine. Therefore, the absence of urobilinogen in the urine is the most reliable evidence of total biliary obstruction. While this feature is not absolute, it is nevertheless the most important single criterion of a distinguishing nature. It is accordingly this simple test that is so useful in separating hepatic-cell disease from bile-duct obstruction in cases of choluric jaundice.

Thus the blood pigment alterations may be understood according to whether they are faults of synthesis or faults of degradation. The former include the true and acquired porphyrias. The latter include the disorders characterized by jaundice. The most important types of jaundice are those due to overproduction of pigment (acholuric) and those due to hepatic-cell disease or biliary obstruction (choluric). Between the latter the presence or absence of urobilinogen in the urine is a most useful bit of information. In this way the spectrum of blood pigment disorders may be understood in rudimentary form as alterations within a single conceptual unit.

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Treatment of Vaginitis with a Broad-spectrum Antimicrobial Preparation—AVC Cream

JAMES C. WALSH, M.D.,* JOSEPH B. SHEFFERY, M.D.,† AND THOMAS A. WILSON, M.D.*

VAGINITIS of bacterial or parasitic origin represents a problem for both patient and physician. For the patient it involves discomfort and an interference with normal behavioral patterns. For the physician it necessitates the treatment of a condition which may be caused by one of a variety of organisms responding to different therapeutic agents, which may represent a mixture of pathogens, or which may begin as one type of infection and, when that is treated, change to another through overgrowth of a second pathogenic organism.

Vaginitis is most commonly due to bacteria (nonspecific), *Trichomonas vaginalis*, or *Candida albicans*. Although temporary symptomatic relief can usually be obtained, eradication of the infecting organisms is more difficult; the disease is characterized by frequent recurrence at menses, particularly in the case of Trichomonas and Candida.[1] Treatment is further complicated by the fact that mixed infections may occur. Concurrent appearance of monilia has been reported in 10 to 15 per cent of trichomonal infections.[2, 3] In many instances monilia has appeared following institution of therapy in patients being treated for initially pure trichomonal infections.[3, 4, 5] The reason for this is not clear but is presumably related to the dynamics of floral equilibrium in the vaginal tract.

These considerations suggest the desirability of treating vaginitis not only with an effective antimicrobial preparation but with one which has a sufficiently broad spectrum to be active against all the infectious organisms commonly involved either singly or collectively. The present study deals with the evaluation of such a preparation in a group of patients in a private obstetrics and gynecology practice.

Materials and Methods

Fifty-two patients were originally admitted to the study. They were between the ages of 15 and 45 years, generally healthy, normally menstruating, and hormonally in balance. All presented evidence of vaginitis in the form of vaginal discharge and irritation. Microbiologic diagnosis was made by examination of a wet-mount preparation, culture on Nickerson's medium, and acridine orange stain.

Patients were given a preparation‡ containing sulfanilamide 15.0 per cent, aminacrine hydrochloride 0.2 per cent, and allantoin 2.0 per cent in a cream base buffered to pH 4.5, and instructed to use 1 applicatorful (about 5 Gm.) nightly on retiring. Period of treatment was set at 21 days. The patients were requested to refrain from the use of internal tampons and to continue medication if menstruation occurred during the treatment period.

Patients were examined again from 2 to 5 days after completion of therapy and subjective and objective findings recorded. Microbiologic examinations were repeated in order to determine the effect of the treatment on the infecting organism. A final visit was made within 5 days after the next menstrual period following treatment. At this time another physical examination and microbiologic evaluation were made.

Results

Of the original 52 patients, 5 were lost to follow-up, so that 47 completed the study. Of these 47 patients 15 were diagnosed as having monilial infections, 6 trichomonal, and 7 mixed monilial and trichomonal. The remaining 19 had nonspecific bacterial vaginitis.

Results based on microbiologic evaluation are presented in table 1. The diagnosis of nonspecific vaginitis was established by exclusion, and final evaluation of this condition was made only in terms of clinical response. In a total of 22 cases of monilial infection (7 of which were associated with Trichomonas) 20, or 91 per cent, were cured at the end of treatment. In only 3 cases did the condition reappear after menses. Cures were achieved in 9 of the 13 patients with trichomonal infection (7 associated with monilia), for a cure rate of 69 per cent. There were no recurrences of trichomonal infection following menses.

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† Clinical Associate Professor of Obstetrics and Gynecology, Georgetown University School of Medicine.

‡ AVC Cream, a product of The National Drug Company, Philadelphia, Pa.

TABLE 1: Microbiologic Cure Rates with AVC

Infecting Organism	Cures/Total	
	Before Menses	After Menses
Monilia	14/15	11/14*
Trichomonas	4/6	4/5*
Both Monilia and Trichomonas	6/7	6/7
	5/7	5/7

* One patient did not return after menses.

The clinical cure rates, based on evaluation of symptomatic relief and disappearance of gross evidence of vaginitis, are presented in table 2.

TABLE 2: Clinical Cure Rates with AVC

Infecting Organism	Cures/Total					
	Before Menses			After Menses		
	Complete	Partial	Failure	Complete	Partial	Failure
Monilia	12/15	3/15	0/15	12/14*	2/14	0/14
Trichomonas	4/6	0/6	2/6	4/5*	0/5	1/5
Monilia and Trichomonas	5/7†	0/7	2/7	5/7	0/7	2/7
Bacterial, nonspecific	17/19	1/19	1/19	14/15‡	1/15	0/15

* One patient did not return after menses.

† Clinical failures coincide with uncured trichomonal infection.

‡ Four patients not evaluated.

Complete cure was obtained in 17 of 19 patients with nonspecific vaginitis, and partial cure in 1. Because 4 patients did not return for postmenstrual evaluation, no conclusions can be drawn about recurrence. Clinical cure rates for Trichomonas and Monilia are in good agreement with the microbiologic results. No side-effects of any type were observed.

Discussion

Although it is difficult to generalize from the study of a relatively small number of patients, some interesting observations emerge from the results reported above. Highly satisfactory therapeutic effects were obtained with the agent used in all types of pure and mixed infections identified in the study. The effectiveness against monilial and trichomonal infections is of particular interest, since these conditions are often treated by agents which do not have the advantage of broad-spectrum activity. The results confirm reports of other investigators.[6, 7, 8] In consideration of the small number of failures observed, it should be pointed out that, within the limits of the study, treatment schedules were not tailored to individual patients. In order to have meaningful comparative results we thought it necessary to employ a standard treatment schedule. This was set in the original protocol as daily application for a period of 21 days. In actual practice, average duration of treatment was 20.7 days, with an average of 2.4 days missed during this period. It is thus conceivable that longer treatment periods might have converted some of the failures into cures.

The number of recurrences of infection after menses was less than would have been expected. In view of the small number of patients involved, and the lack of specific data on the incidence of such recurrences, this can be offered only as an observation.

No secondary infection due to overgrowth developed during the course of treatment in any of the patients; this would not in any case have been expected with the use of a broad-spectrum antimicrobial preparation. Aside from this factor the justification for the use of such a broad-spectrum agent would appear from an analysis of the diversity of infecting organisms involved in vaginitis in a cross-section of an obstetrics-gynecology practice. Division in the patient group was: bacteria, 40 per cent; Monilia, 32 per cent; Trichomonas, 13 per cent; and mixed Trichomonas and Monilia, 15 per cent.

Summary

Forty-seven patients with vaginitis of varying etiology were treated with a broad-spectrum anti-infective preparation containing sulfanilamide, aminacrine hydrochloride, and allantoin. Initial microbiologic cure rate in the group, including trichomonal, monilial and mixed trichomonal-monilial infections, was 83 per cent. Initial clinical cure rate in the entire group was 81 per cent. Rate of recurrence following menses was low. No side-effects were encountered.

(*Continued on page 364*)

Automatic Spirographic Analysis

Results of the Demonstration Held at COPD-68

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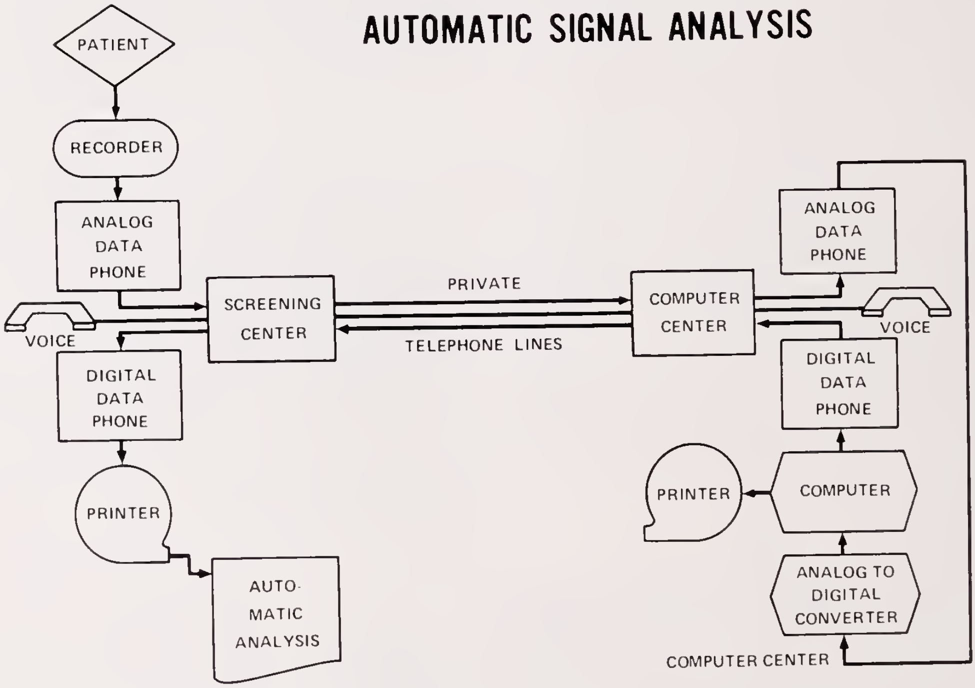
The Medical Systems Development Laboratory (MSDL) of the U. S. Public Health Service has applied computer techniques to the analysis of the forced expiratory spirogram (FES). In conjunction with the D. C. Health Department the system was demonstrated at COPD-1968, a Symposium on Chronic Obstructive Pulmonary Disease, sponsored by the D. C. Thoracic Society, January 10 and 11, 1968. Local physicians and paramedical personnel were invited to participate in the demonstration by performing forced exhalations.

The system (figure 1) requires a spirometer with an electrical output. The signal is recorded on a specially constructed data acquisition cart§ available commercially. A coding device enters the recording location of the test, the subject's identification number, and pertinent demographic data on paper and FM magnetic tape. The same electrical signal can be entered into a data transmitting telephone and carried via ordinary telephone lines to the computer facility where the FES is processed. If the results are not required immediately the FM tapes can be mailed to the processing location. The interpreted measurements and calculations are returned to the recording site either by mail or over phone lines to a teletype which prints the final report.


AUTOMATIC SIGNAL ANALYSIS
PATIENT
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SCREENING CENTER
PRIVATE
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AUTOMATIC ANALYSIS
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ANALOG DATA PHONE
VOICE
DIGITAL DATA PHONE
PRINTER
COMPUTER
ANALOG TO DIGITAL CONVERTER
COMPUTER CENTER

FIG 1. Schematic representation of automatic signal analysis.

The printout includes the demographic and identification data, measurements of the forced vital capacity (expressed in milliliters and as a percentage of the volume predicted by the Kory formula[1]), forced expiratory volumes at selected time intervals (both in milliliters and as percentages of the performed forced vital capacity), and flow rates at selected points along the expiratory curve. The automated calculations[2] have been shown to be precise and accurate,[3] clinically applicable,[4, 5] and less variable than manual methods.[6] Alteration of the signal by telephone transmission is less than observer variation introduced by hand measurement.[7]

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§ Dat-EK Model 6509.

Comment

Two and one-half hours were required for the demonstration, which encompassed the recording, transmission, measurement, interpretation, and printing of the complete records. The results are shown in table 1. Twenty-eight individuals volunteered, 4 of whom were female. The average age of the participants was 45 years. Overall, 35 per cent of the records were abnormal. On an age basis, 44 per cent of those aged 40 and above and 20 per cent of those under 40 were below expected norms. No clinical information was available and one ought not reach a diagnosis based solely on a spirometric recording. It is known that 35 to 58 per cent [8,9] of those records considered to be abnormal by screening techniques are later found to be normal when follow-up validation procedures are performed.

TABLE 1: Number and Per cent of Volunteers with Abnormal Spirograms by Age

Age (yrs.)	Average Age (yrs.)	No.	Abnormal Spirograms	
			No.	Per cent
>40	50.3	16	7	43.7
<40	30.8	10	2	20.0
TOTALS*....	45.0	26	9	34.6

* Total excludes 1 record lost in transmission and 1 record considered uninterpretable.

At the present time the MSDL processes spirographic signals routinely from the Preventive Medicine Clinic at the U. S. Public Health Service Hospital, San Francisco, California, the Milwaukee City Health Department, and the Ohio State Department of Health, as well as epidemiologic surveys under the auspices of the National Health Survey and Harvard School of Public Health.

This demonstration illustrates the type of service that can be performed with modern communications and computer techniques. No physician need be involved in the recording or analysis of these records.

The computer releases valuable time of the physician from routine laboratory work and allows him more time to perform his primary duty, the direct care of the ill.

The computer is also programmed to measure and interpret other medical signals, such as the electrocardiogram and the electroencephalogram.

Our especial thanks are extended to the staffs of the Medical Society of the District of Columbia and the Medical Systems Development Laboratory, U. S. Public Health Service, for their obvious interest and attention.

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How the Emergency Room of a General Hospital Serves the Health Needs of Children from Low-income Families

Profile of a Pilot Study Based on Six Months' Observation

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THE PUBLIC is becoming more demanding in its search for readily available convenient medical services. This demand is forcing a changing role upon the function of the hospital emergency room. In an effort to update our service and make it more responsive to community needs we have surveyed our pediatric emergency room service.

Freedmen's Hospital, a 500-bed general hospital, is the main teaching facility for Howard University College of Medicine. It is located in a low-income residential area, where the population is predominately Negro, in northwest Washington, D. C. The hospital has 50 beds and outpatient clinics for children as well as a busy pediatric emergency service.§ Infants and children under 14 years of age are accepted for pediatric care. The pediatric emergency service is staffed by residents from the Pediatric Service of the hospital and is supervised by medical officers and members of the attending staff of the Department of Pediatrics of the Howard University College of Medicine.

A previous survey of the emergency service performed in the summer of 1965 revealed that 48 per cent of pediatric patients were under 3 years of age; 35 per cent lived in the vicinity of the hospital.[1] These observations are extended in this report.

Results

For the present survey all patient visits from January 1, 1967, to June 30, 1967, a total of 2,811, were tabulated. The demand on the emergency room service is increasing each year, as shown by table 1. This is to be expected since the U. S. Public Health Service has projected an increasing use of hospitals for the decade 1960–70. Of 8 per cent of hospital admissions 18 per cent were outpatient visits and 79 per cent were emergency department cases.[2]

TABLE 1: Visits Made by Patients in the Pediatrics Emergency Room During the 10-year Period 1956–1966

Year	Total No. of Visits
1956	1,979
1957	2,279
1958	4,564
1959	4,340
1960	5,236
1961	4,873
1962	5,406
1963	5,753
1964	6,170
1965	7,736
1966	8,968

Most pediatric patients need emergency room care in the afternoon (40 per cent) or evening (30 per cent), as shown in table 2. This is undoubtedly related to problems of transportation, child care for siblings, and the lack of availability of working parents during the earlier hours of the day.

The frequency of disease, particularly infections in the younger age-groups, is reflected by the fact that 50 per cent of the patients were under 2 years of age.

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§ Freedmen's Hospital is located in the geographic area which comprises census tracts 34 area 5 where the annual median family income based on statistics for the year 1960 was $5,000.

TABLE 2: Selected Statistics on 2,811 Children Seen in the Pediatric Emergency Room from January 1, 1967 to June 30, 1967

	No. of Patient Visits	Percentage
1. Time registered:		
6 a.m. to 12 noon	540	21
12:05 p.m. to 6 p.m.	1,224	40
6:05 p.m. to 12 a.m.	896	33
12:05 a.m. to 6 a.m.	151	6
2. Ages:		
Birth to 2 years	1,446	50
2 years to 4 years	624	23
4 years to 6 years	217	7
6 years to 14 years	524	20
3. Sex:		
Male	1,574	55
Female	1,237	45
4. Address:		
Northeast, Washington, D.C.	575	20
Northwest, Washington, D.C.	1,743	63
Southeast, Washington, D.C.	307	11
Southwest, Washington, D.C.	116	4
Maryland	70	2
5. Race:		
Negro	2,764	99
White	47	1
6. Accompanied by:		
Mother only	2,432	87
Father only	171	6
Parents	107	4
Guardian	88	3
7. Referred by private physician	2,644	94
Not referred by private physician	167	6
8. Disposition:		
Child sent home	1,526	55.2
Referred to clinics of this Hospital	884	34.0
Admitted to inpatient service of this Hospital	145	5.2
Sent back to private physician	152	5.4
Transferred to D. C. General Hospital	4	0.2
Antibiotics prescribed	882	31
Antibiotics not prescribed	1,929	69

Sixty-three per cent of the patients resided in the section of Washington where the hospital is located. Only 2 per cent came from Maryland. Since Freedmen's Hospital traditionally has served Negro citizens, as expected 99 per cent of the children were colored. In 87 per cent of the cases the child was brought to the hospital by the mother.

The fact that 94 per cent of the children in the present survey had no private physician reflects the mobility of the population as well as their low economic status. Of the 6 per cent who had a private physician, only a few had been referred to the hospital by him. The rest were apparently loath to call upon the doctor's services outside of his usual office hours.

Diagnoses are listed in table 3 of conditions which could be life-threatening (trauma, convulsive disorders, acute abdomen, ingestions of poisons, burns, and acute cardiac problems) and constitute only 6.7 per cent of total visits. Conditions which could be treated in the regular outpatient clinic (respiratory-tract infections, skin disorders, and eye, ear, nose and throat conditions) were the main reason for "emergency" room visits. Another indication of the nonemergency nature of the majority of these cases is

TABLE 3: Diagnostic Categories During Period of Observation (January 1, 1967 to June 30, 1967)

Selected Diagnostic Categories	No. of Patient Visits
1. Respiratory tract infection (acute bronchitis, tonsillopharyngitis, acute epiglottitis, bronchopneumonia, upper respiratory tract infection)	1,268
2. Skin disorders (impetigo, urticaria, eczema, contact dermatitis, etc.)	239
3. Ear, nose and dental problems	221
4. Gastroenteritis	217
5. Viral exanthemata	140
6. Bronchial asthma	128
7. Trauma (concussions, contusions, lacerations, etc.)	95
8. Genitourinary problems (inguinal hernias, urinary tract infections, phimoses, etc.)	76
9. Eye problems	60
10. Normal children (no disease)	51
11. Ingestions	47
12. Convulsive disorders	25
13. Orthopedic problems	19
14. Gynecological problems (vaginitis, rape, etc.)	17
15. Foreign bodies	15
16. Endocrine problems (diabetes mellitus, hyperthyroidism, obesity, etc.)	13
17. Acute abdomen (acute appendicitis, incarcerated hernias, intussusception, etc.)	12
18. Anxiety reactions	12
19. Sickle-cell and thalassemia crises	12
20. Feeding problems	11
21. Cardiac problems (rheumatic heart disease, congenital heart disease, etc.)	7
22. Tumors	5
23. Fever of unknown origin	5
24. Muscle spasm and torticollis	4
25. Burns	3
26. Dead on arrival	2
27. Esophageal stricture	1

that 55.2 per cent were sent home, 34.0 per cent were referred to specialty clinics, and a mere 5.2 per cent were hospitalized. These findings are in accord with an editorial in *News of Metropolitan Washington*[3] which states that only 10 to 15 per cent of emergency-room visits are true emergencies in a life-threatening sense.

It is apparent that, in addition to serving as an accident room, our pediatric emergency room functions as an after-hours "private" physician's office, a general pediatric clinic, and a screening facility for various subspecialty clinics.

The U. S. Children's Bureau has estimated that there will be approximately 91.3 million children in the United States in 1970 and that children will comprise 42.3 per cent of the population. In 1970 the ratio of children to adults will be 87 to 100. This is a 10 per cent increase over the child-to-adult ratio for 1960 and a 37 per cent increase over 1950.[4]

Conclusions

Our observations for the period of study may serve as a forecast to indicate the need for newer and more realistic approaches to the delivery of care for low-income families. The following statement appeared in *Medical World News*: "For decades the so-called emergency facilities of most hospitals have consisted only of accident rooms, poorly equipped, inadequately manned, and ordinarily used for limited numbers of seriously ill patients."[5]

There is an expanding utilization of the emergency room to meet the needs for the ambulatory care of children whose families occupy the depressed areas of the inner city.

The staff working in this area of the hospital has become the "family pediatrician" for the patients who use the service.

Since 50 per cent of the children are under 2 years of age, the facilities in the emergency area should include supplies, drugs, equipment, and personnel which can satisfy the peculiar needs of infants and very young children. This is important in view of the current excessive infant mortality among the nonwhite population in Washington, D. C.

In view of the nonemergency nature of disorders of the overwhelming majority of the children seen in the emergency room there is need for initial "screening" procedures in order to separate those with mild illnesses from those who represent true emergencies and hence require special procedures immediately.

After initial "screening" children who do not require emergency attention could best be handled in afternoon and night outpatient clinics where they could be given the pediatric care which would be indicated based upon environmental circumstances and the physician's findings.

Demographic predictions and projections indicate that hospitals which are in the planning stages should seek to provide ample and expandable facilities for ambulatory patients, particularly for children, in order to best serve the health needs of the community.

The emergency department of Freedmen's Hospital is undergoing renovation and modernization in order to improve its services both for adults and children.

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Treatment of Vaginitis—Walsh, Sheffery and Wilson

(*Continued from page 359*)

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Metastatic Carcinoma of the Ciliary Body*

Report of a Case

PEDRO I. MATIAS, M.D.† AND BRUCE I. SHNIDER, M.D., F.A.C.P.‡

QUITE FREQUENTLY carcinomas metastasize to various visceral and skeletal organs. This is especially true in cases of carcinoma of the lung, breast, and various gastrointestinal malignant lesions. Some types of malignant disease are notorious in their great ability to metastasize by hematogenous spread, such as malignant melanoma.

The incidence of ocular involvement in metastases, however, is not often seen and is more infrequent when the part involved is the anterior chamber of the eye. It becomes very rare when the primary carcinoma is esophageal in origin. It is the purpose of this brief clinical note to present an unusual pattern of metastases in a carcinoma of the esophagus and to review some pertinent information from the medical literature.

Report of Case

A 56-year-old white man was admitted to the District of Columbia General Hospital on August 15, 1966 for evaluation of "red eye." He stated that he had been in good health until 3 weeks prior to admission when he noted that his right eye had become red. He was seen in the Eye Clinic where a diagnosis of acute iritis was made and was admitted because of a suspicion of malignancy due to a history of a 50-pound weight loss of several weeks' duration, dyspnea on exertion of 1 week's duration, and dysphagia and a sensation of food "sticking" at the midsternal level. He denied any history of orthopnea, melena, hematemesis, or chest pain.

The past history was not remarkable except for poliomyelitis at 2 years of age, with residual right-leg atrophy and foot drop. One brother had died with cancer and one sister had congestive heart failure due to arteriosclerotic heart disease.

The patient was a painter who had smoked 20 cigarettes a day for many years and drank ½ pint of whiskey a day. For 3 months he had had urinary hesitancy and nocturia.

The patient was in no acute distress; he had a right foot drop and an erythematous right eye. Visual acuity was 20/70, tonometric pressure was 25.8 mm. Hg (left 13.1 mm. Hg) with 7.5 Gm. weight, the pupils were round, equal (1 mm.) and nonreactive. There was a 1.0 mm. bleb-like projection at the 8 o'clock position of the limbus. Slit lamp examination showed the cornea to be clear; the anterior chamber revealed 1+ cells and 2+ flare; there were white porouslike lumps in the periphery of the iris from 6 to 8 o'clock. The iris was distorted and congested. The lens showed nuclear sclerosis. The fundi were not seen. There were no lymph nodes palpable in the neck. There were flatness and diminished breath sounds over the right lower lobe of the lungs. Abdominal examination revealed no organomegaly, and the bowel sounds were active. The extremities showed good peripheral pulses. The right leg was atrophic.

Urinalysis showed albuminuria of 30 mg. per cent, 90–100 leukocytes, and 4+ bacteriuria. Urine culture revealed paracolon, Aerobacter and Escherichia species, on 3 occasions, for which the patient was given appropriate antibiotic therapy. The hematocrit ranged between 24 and 28 per cent, and the blood urea nitrogen was between 33 and 50 mg. per cent. Serum alkaline phosphatase was 11.6 King-Armstrong units; calcium was 9.8 mg., and phosphorus 4.0 mg. per 100 ml. Intermediate strength PPD was negative. A chest X-ray film revealed multiple calcified hilar nodes indicative of an old granulomatous process. Upper gastrointestinal X-ray examination showed an irregular, partially obstructing lesion of the esophagus at T-5 level extending for about 7 cm. (figure 1). Esophagoscopy revealed a fungating, slightly hemorrhagic lesion at 23 cm. from the incisors, and biopsy showed a well-differentiated squamous-cell carcinoma.

Five days after admission the patient was transferred to the Georgetown Medical Division where examination revealed essentially the same findings. He now claimed that vision in the right eye was totally absent.

The growth of the eye lesion was rapid, and by the tenth day hyphema had developed. On the fourteenth day satellite lesions had developed in the conjunctiva, and the anterior chamber was filled (figure 2). On the following day excisional biopsy of the conjunctival tumor was done and revealed well-differentiated squamous-cell carcinoma, similar to the esophageal biopsy.

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A gastrostomy was performed on September 7, 1966 because of progressive dysphagia. The patient was discharged shortly thereafter to undergo radiotherapy in another hospital. He was readmitted to a surgical ward on September 16, 1966, because of leakage around the gastrostomy tube. The clinical course was progressively downhill. Painless hematuria also developed while he was in the hospital, but because of his poor condition no further diagnostic studies were carried out, and he expired on September 29, 1966. An autopsy was not performed.

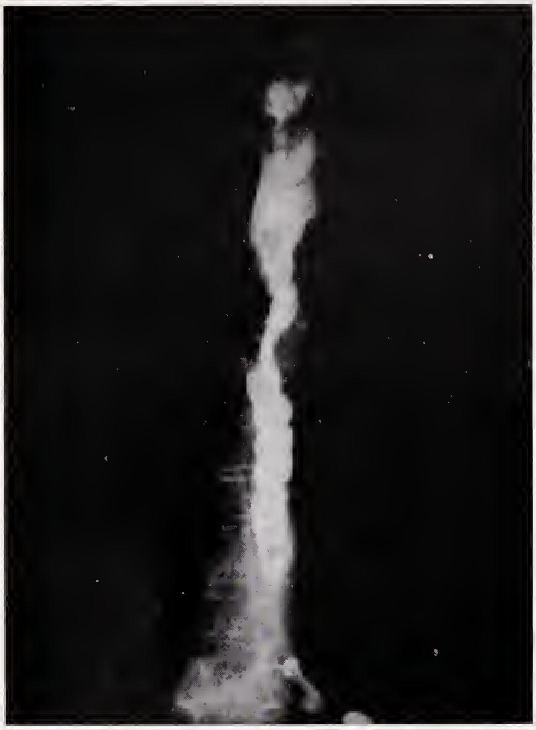

FIG. 1. Barium swallow showing partial obstruction at middle third of esophagus.

Discussion

The incidence of ocular metastases is very low. In 1963 Titche[1] reported a case of ocular metastases from bronchogenic carcinoma. He referred to Duke-Elder who stated that carcinomata are the most common tumors to metastasize to the eye, probably because the metastases are bloodborne.

In 1961 Mork[2] reviewed the incidence of malignant neoplasms of the eye. Of 273 cases of malignant neoplasms of the eye in the Cancer Registry of Norway from 1953 to 1960, 81 per cent were melanomas, 11 per cent were retinoblastomas, and 8 per cent were other tumors. This report, however, did not show the incidence of ocular involvement due to metastases. In 1965 Güthert et al[3] examined the posterior part of the eyeball for metastases in 1,000 autopsies of patients with malignant tumors. There were 853 carcinomas, 91 sarcomas, and 56 neuroectodermal tumors. Metastases in the eye were found in only 5 cases, and 4 of these were from carcinomas and 1 from a sympathicogonioma, all involving the choroid.

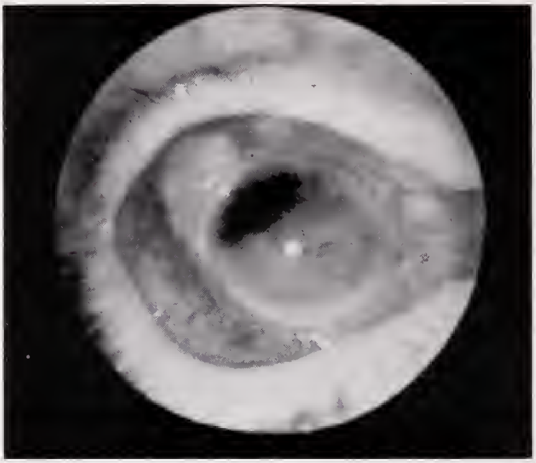

FIG. 2. The right eye on day 14.

Roussel et al[4] reported a case of bronchogenic carcinoma with metastases to the ciliary body and reviewed 18 case reports during the years 1900 to 1963. A more extensive review was done by Thomas et al.[5] From 1890 to 1964 there were 74 case reports of metastatic cancer to the anterior uvea, including a case of bronchogenic carcinoma which they presented. By far the most frequent primary tumors are of the breast and lung. Only 1 case of carcinoma of the esophagus was referred to in this review; thus the present case is the second reported case of esophageal carcinoma with metastatic involvement of the anterior uvea.

Attesting to the rarity of ocular involvement in esophageal carcinoma, the Armed Forces Institute of Pathology Fascicle on Tumors of the Esophagus[6] makes no mention of metastases to the eye. A careful study by Klein in 1927 showed the usual mode of metastases of esophageal carcinoma.[6] Of 264 autopsy cases, no metastases were found in 31 per cent. There was direct invasion of the trachea in 52 cases, of the bronchi in 32 cases, and of the mediastinum in 33 cases. The aorta was penetrated by the tumor 7 times. The regional and distal lymph nodes received metastases more frequently than any other site since the esophagus is drained by numerous lymphatic systems, which include the para-esophageal nodes, the paratracheal nodes, the parabronchial nodes, the posterior mediastinal nodes, the paracardiac nodes, and the deep cervical nodes. The pattern of metastases from carcinoma of the esophagus was studied by Neuberger.[7] By far the most frequent site of metastases is the regional lymph nodes. The liver and the lymphatic channels in the esophagus are the next most frequent sites (table 1).

TABLE 1: Frequency of Metastases Found at Autopsy in 192 of 301 Patients*

Site	No. of Patients	Per cent
Lymph node	153	50.00
Liver	62	20.50
Lymphatic channels in esophagus	37	12.30
Lungs	20	6.60
Kidney	19	6.30
Bone	10	3.30
Pleura	8	2.60
Adrenal gland	8	2.60
Thyroid gland	6	2.00
Myocardium	5	1.90
Omentum	5	1.90
Pancreas	4	1.30
Spleen	3	1.00
Peritoneum	3	1.00
Intestine	3	1.00
Pericardium	3	1.00
Stomach	2	0.66
Tongue	1	0.33
Epiglottis	1	0.33

* Based on data of Neuberger.[7]

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Safety Tips for Fishermen

The American Red Cross program, with the aim of getting more and more Americans trained and knowledgeable in the fields of swimming, lifesaving, first aid and accident prevention, lists the following safety tips for followers of the piscatorial pastime:

1. All fishermen—experts and novices alike—should know how to swim, know basic first aid, and have a first aid kit with them.
2. Listen to weather forecasts so you can dress properly.
3. Since fish hooks sometimes "catch" anglers as well as fish, carry needlenose pliers, wire cutters and pocket knife in your tackle box. Always keep hooks in covered containers.
4. In casting make certain you have room to do so without hooking someone else. Use an overhead cast when in a boat with others, and always look before casting.
5. When going on a fishing trip alone, be sure to leave word where you will be and when you plan to return. It's much safer, however, to fish with a companion.
6. When fishing in water where there are swimmers, water skiers, and skin and scuba divers, be careful to keep your boat and your fish lines in open areas.
7. Wear thick-soled boots or hiking shoes in densely wooded or marshy areas but do not wear waders while in a boat. Barefoot fishermen sometimes step on broken glass, nails and other debris resulting in serious injuries.
8. Wade cautiously to avoid slipping on unseen rocks or stepping suddenly into deep water.
9. Learn how to hold a fish properly while extracting a hook. Many species have sharp teeth or fins that can cause deep hand wounds.
10. If you snag yourself deeply with a hook, cut off the line, bandage the wound, and head for the nearest doctor or hospital. Don't risk further injury by trying to remove a deeply-imbedded hook.
11. In snake-infested country, watch where you step, what you touch, and where you sit. Wear high boots because half of all snake bites are below mid-calf.
12. When fishing in channels or rivers, never tie your boat to channel buoys. It is illegal. These are traffic lane markers and must be kept clear at all times.
13. Never stand up in a boat or canoe when fishing, and be sure to have a life preserver on board for every passenger.

The Medical Expert Witness
vs.
The Power of the Printed Page

MAURICE H. HERZMARK, M.D.*

Most medical specialists have published articles or books pertaining to their specialties. These contributions to medical literature tend to establish them as authorities. Thus, they are often called upon to appear in court as expert medical witnesses. Lawyers, learning the identity of a physician whom they need to cross-examine, find it expedient to repair to a medical library to read up on any articles or books published by the expert. A careful note is made of any statement made in these writings which may be used to challenge the testimony given on the witness stand.

A physician who reports his experiences or research may from time to time alter his views as he improves his knowledge, but the attorney, knowing that the court cannot keep up with medical science, may select any published statement, regardless of the time of its publication, and use it to challenge the expert, quoting directly from the article or book. If the doctor tries to explain that the material is out of date, he is quickly cut off with the admonition: "Just answer the question. Did you write this?" If the doctor tries to explain that it is no longer valid, he creates the impression that he is trying to evade the issue and presents an undesirable impression with the court. Confronted with such a situation, the doctor's best approach is to appeal to the judge to permit an explanation before answering. Not every judge will grant this, but it is worth the try. Sometimes sentences or paragraphs are quoted out of context and the witness is requested to comment on the correctness of those statements without regard to mitigating assertions which may precede or follow. The scientifically-minded physician is often at a great disadvantage when testifying because the rules of court procedure force him to answer questions which, though truthful, produce a wrong impression.

To illustrate this technique, here is a brief résumé of a recently tried case. A surgeon performed an arthrotomy on a badly deformed elbow caused by tuberculosis incurred in childhood. Previously another surgeon had attempted to mobilize the joint by inserting fascia lata, but the elbow became partially ankylosed in flexion. Sixteen years later the joint was opened, a large mass of debris was removed, and the greatly enlarged olecranon was excised to permit extension to about 160 degrees. The lower end of the humerus was deformed so that the landmarks were not present. There was no ulnar groove and no ulnar nerve was visible, it being encased in a dense scar. The bone was exposed by subperiosteal dissection, and all soft tissues were protected by retractors. The wound healed, the patient obtained the desired extension, and he returned home, driving his car a distance of 250 miles without stopping. Following this the surgeon received a letter in glowing tribute to the success of the operation. But, although the patient received payment for the services rendered by the surgeon from an insurance company, he did not remit anything to the physician. Nor did he complain. He simply ignored all bills. When a credit company finally brought suit on behalf of the surgeon, a counterclaim was filed citing damage due to cutting of the ulnar nerve. During postoperative examinations there was no evidence of injury to any of the nerves. The nurses' notes, the surgeon's notes, and a postoperative photograph showed no evidence of ulnar palsy. The case came to trial.

In the operative notes no mention was made of the ulnar nerve because it was not in the way and had not been isolated, since, enclosed in the dense scar tissue, it was much better protected than by a tape or tube. But, textbooks describing operations on the elbow recommend isolating the ulnar nerve and retracting it with a tape or tube. This is applicable when the nerve is in its normal position and not encased in a fibrotic mass. The attorney for the patient knew that the textbooks described the retraction procedure so he ignored the fact that the case was not the kind described in textbooks; not wishing to risk an explanation he inquired: "Doctor, isn't it a fact that authors on surgical procedure consider it good practice to isolate the ulnar nerve?"

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WITNESS: "Yes, if the nerve is in danger."

LAWYER: "Doctor, is it bad practice to cut the ulnar nerve?"

WITNESS: "Of course, but the nerve was not. . . .

LAWYER: "I merely asked you if it is bad practice to cut the nerve, not anything else."

JUDGE: "He answered the question."

Note the subtlety of this question. The effect was to plant in the minds of the jurors the notion that the surgeon did cut the nerve.

The only way to answer such a question is to state first that the direct testimony showed that the nerve was not cut and that the question was misleading because the answer is obvious and irrelevant.

The lawyer relied on textbooks, overlooking the fact that experienced surgeons must be guided by the pathology found and use their own best judgment. Yet the question as it was put made an impression on some of the jurors, resulting in a large verdict for the patient.

If a witness, however qualified, has ever published anything, even if not related to the case at hand, the attorney may try to use it to his advantage. In a recent case when I was a medical witness the defense attorney started his cross-examination with the following:

LAWYER: "Doctor, isn't it true that you wrote an article for *Medical Economics* entitled 'Tips on Testifying—How to Get Through to the Jury'?"

DOCTOR: "Yes, to prepare my medical colleagues for questions by tricky lawyers such as you seem to be."

Of course he moved to strike out the last part of my answer as not relevant, but the jury got the message, and the attempt to deride me lost its force.

Outstanding specialists often write brief papers calling attention to certain dangers and urge some change or improvement. A urologist attached to a large hospital, noting that blood transfusions could be a means of transmitting syphilis, wrote a brief essay on the subject and urged that persons whose blood was to be used for transfusion be given either a Kahn or Wassermann test. Many years after this was published he became Chief of Urology at Mt. Sinai Hospital in New York and was also elected President of the New York Academy of Medicine. In this capacity he was called upon to defend a surgeon who had used the blood of a volunteer donor to give to a very debilitated child. The donor was a close friend of the father, a healthy-appearing young man, who was a fellow musician in a symphony orchestra. He went to the hospital, his blood was typed and cross-matched, but no Kahn or Wassermann test was ordered or done. Regrettably, this man had syphilis, and after the blood was transfused the child developed the early manifestations of the disease. By a process of elimination the donor became suspect, but he refused to submit to any tests, claiming he had done his share by giving his blood. Despite treatment the child failed to respond and remained "Wassermann-fast." The father now brought a suit, enjoining the hospital and the surgeon in a malpractice action. The attorney obtained a court order and had the donor apprehended and forced to submit to tests. When these proved positive he admitted that he had had the acute manifestations some years previous and had had 1 treatment at a public health clinic. He claimed that when the acute symptoms subsided he thought he was cured. The New York Medical Society asked the prominent urologist to defend the surgeon on the grounds that since he was not a hematologist he was not bound by the same rigid standards applicable to a highly specialized field. But the lawyer for the plaintiff found the article which warned of the danger and recommended that blood be tested before transfusing it no matter whose it is or who is giving it. When the urologist was confronted with this the judge called the litigants to his chambers and suggested a settlement in favor of the child. The carrier was required to pay a fairly large sum. If the defending witness had not been confronted with his own publication the defense might have been valid.

Lawyers confronted by eminent authorities will sometimes resort to strategy in preparing the cross-examination. The following case illustrates how a very distinguished physician became the victim of an unusual scheme. Some years ago a wealthy business executive developed severe pulmonary tuberculosis with cavity formation and frequent hemorrhages. His physician advised that he go to a special sanatorium. He was 57 years of age and was covered by a policy which provided $2,000 per month if incapacitated, but if permanently incapacitated before the age of 60 he could receive the benefits for life, with waiver of premium.

After 2 years the cavity had healed and the hemorrhages had ceased, and the physicians at the sanatorium allowed him to leave. Returning to

New York, his own lung specialist permitted very limited activity, explaining that the healed cavity was near a bronchus and that exertion could activate the disease. Now, approaching the critical age of 60, he was required to present himself for an examination by a physician for the carrier. X-ray films and other tests indicated that the insured was no longer completely incapacitated, and thus the benefits were discontinued. The executive thought that this was unfair because he could not return to his normal occupation or even engage in certain sports which he enjoyed. He retained a lawyer and filed a suit. In preparation for the trial the carriers engaged the most eminent phthisiologist they could find and requested him to examine the plaintiff and appear as a witness. He too examined the man and concurred with the previous physician who found no reason to regard the plaintiff as permanently disabled. The phthisiologist at the time was President of the New York Academy of Medicine, a professor of medicine at a well-known medical school, and the author of a very authoritative work on pulmonary diseases which had gone into 4 editions. The latest edition appeared a few months before the trial. The attorney for the plaintiff knew that the eminent doctor would outweigh the opposing specialist even though he too was very prominent. So he decided on a bit of strategy. He obtained a copy of the professor's latest book and read carefully the portion dealing with prognosis.

As a witness the professor was very impressive. He was a man with a large head covered with a thatch of snow-white hair, a pink complexion, and twinkly blue eyes. He stated that he did not feel that the plaintiff was permanently incapacitated, that he had a healed cavity, no hemorrhages, and to all intents and purposes was an arrested case of pulmonary tuberculosis.

The lawyer arose to cross-examine. He carried with him a large volume, which was actually the latest edition of the professor's book but which was covered with a dust jacket on which in large white letters against a red background was the title, "The Life of Benjamin Franklin." Turning to the page dealing with prognosis and recovery, he said: "Doctor, I have been reading a book which gives some interesting information concerning consumption. I gather from it that once a consumptive, always a consumptive. Would you go along with that idea?"

The expert witness, seeing the title on the dust jacket of the book, thought the lawyer was using Benjamin Franklin as a medical authority, quoting, perhaps, from his famous aphorisms. So he replied, "Certainly not; many cases of tuberculosis are arrested and return to normal life." The lawyer then said, "Being a layman, perhaps I did not paraphrase it properly. I shall read it exactly as it appears in this book: 'It is an unfortunate fact that a very considerable percentage of patients who have taken a cure for tuberculosis and resumed normal activities sooner or later relapse.' Doctor, do you agree with that statement?"

Doctor: "No, it is too broad and too general."

The lawyer then took off the dust jacket and read the title of the book and the name of the author who had just answered the question. Then he said: "The quotation I read is exactly as it appears in your book. Either you have forgotten what you wrote, or someone else wrote it."

It made no difference what was said after that, for the jury was greatly impressed with this verbal unfrocking of an author who could not recognize a quotation from his own book when it appeared to be at variance with his testimony as a witness. The plaintiff won his case.

A medical witness should insist on seeing for himself any material quoted by an attorney. If the doctor had looked at the paragraph which was read to him he would have seen that the subject was amplified and explained. His reply was correct but created a wrong impression, and juries decide by impressions.

"The spoken word flies away like a bird, but that which is written remains like a begotten child—not to be denied."

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In trichomonal vaginitis, most physicians have reported a cure-rate of 95 per cent or more with Flagyl when infected male partners are treated concurrently and when treatment is repeated for occasional refractory infections in women.

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This high rate of cure obtained with Flagyl is unparalleled. Only systemically active Flagyl reaches the hidden reservoirs of reinfection in male and female genitourinary tracts.

Indications: Flagyl is indicated only in the treatment of trichomoniasis in both the male and female.

Contraindications: Pregnancy; disease of the central nervous system; evidence or history of blood dyscrasia.

Precaution: Complete blood cell counts should be made before, during and after therapy, especially if a second course is necessary.

Side effects: Infrequent and minor side effects include nausea, metallic taste and furry tongue. Gastrointestinal disturbances, flushing and headache sometimes occur, especially with concomitant ingestion of alcohol. The taste of alcoholic beverages may be altered. Other effects, all reported in an incidence of less than 1 per cent, are diarrhea, dizziness, vaginal dryness and burning, dry mouth, rash, urticaria, gastritis, drowsiness, insomnia, pruritus, sore tongue, darkened urine, anorexia, vomiting, epigastric distress, dysuria, depression, vertigo, incoordination, ataxia, abdominal cramping, constipation, stomatitis, numbness or paresthesia of an extremity, joint pains, confusion, irritability, weakness, cystitis, pelvic pressure, dyspareunia, fever, polyuria, incontinence, decreased libido, nasal congestion, proctitis and pyuria. Elimination of trichomonads may aggravate candidiasis.

Dosage and Administration: In women: one 250-mg. oral tablet three times daily for ten days. A vaginal insert of 500 mg. is available for local therapy when desired. When used, one vaginal insert should be placed high in the vaginal vault each day for ten days; concurrently two oral tablets should be taken daily.

In men: When trichomonads are demonstrated, one 250-mg. oral tablet twice daily for ten days in conjunction with treatment of his female partner.

Dosage Forms: Oral tablets–250 mg.
Vaginal inserts–500 mg.

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Special Liver-Stomach Concentrate, Lilly (containing Intrinsic Factor)	150	mg.
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Precautions: Anemia is a manifestation that requires appropriate investigation to determine its cause or causes.

In pernicious anemia, the use of folic acid without adequate vitamin B_{12} therapy may result in hematologic remission but neurological progression. Adequate doses of vitamin B_{12} (parenteral, or oral with potent intrinsic factor as in Trinsicon® [hematinic concentrate with intrinsic factor, Lilly]) usually prevent, halt, or improve the neurological changes.

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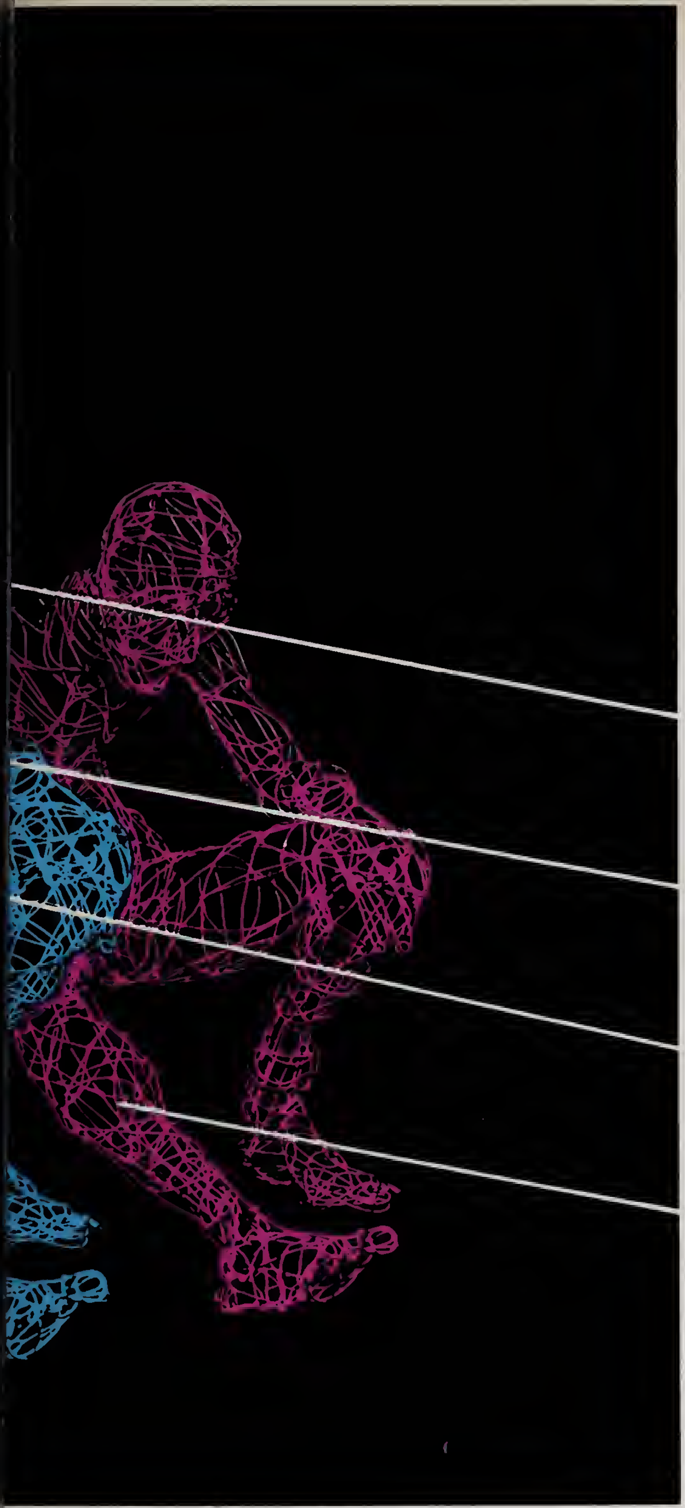

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Ascorbic acid (75 mg.)—augments th conversion of folic acid to its active for and helps iron absorption.

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Adverse Reactions: In rare instances, iron in therapeutic doses produces gastro-intestinal reactions, such as diarrhea or constipation. Reducing the dose and administering it with meals will minimize these effects.

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Dosage: One Pulvule twice a day. (Two Pulvules daily produce a standard response in the average uncomplicated case of pernicious anemia.)

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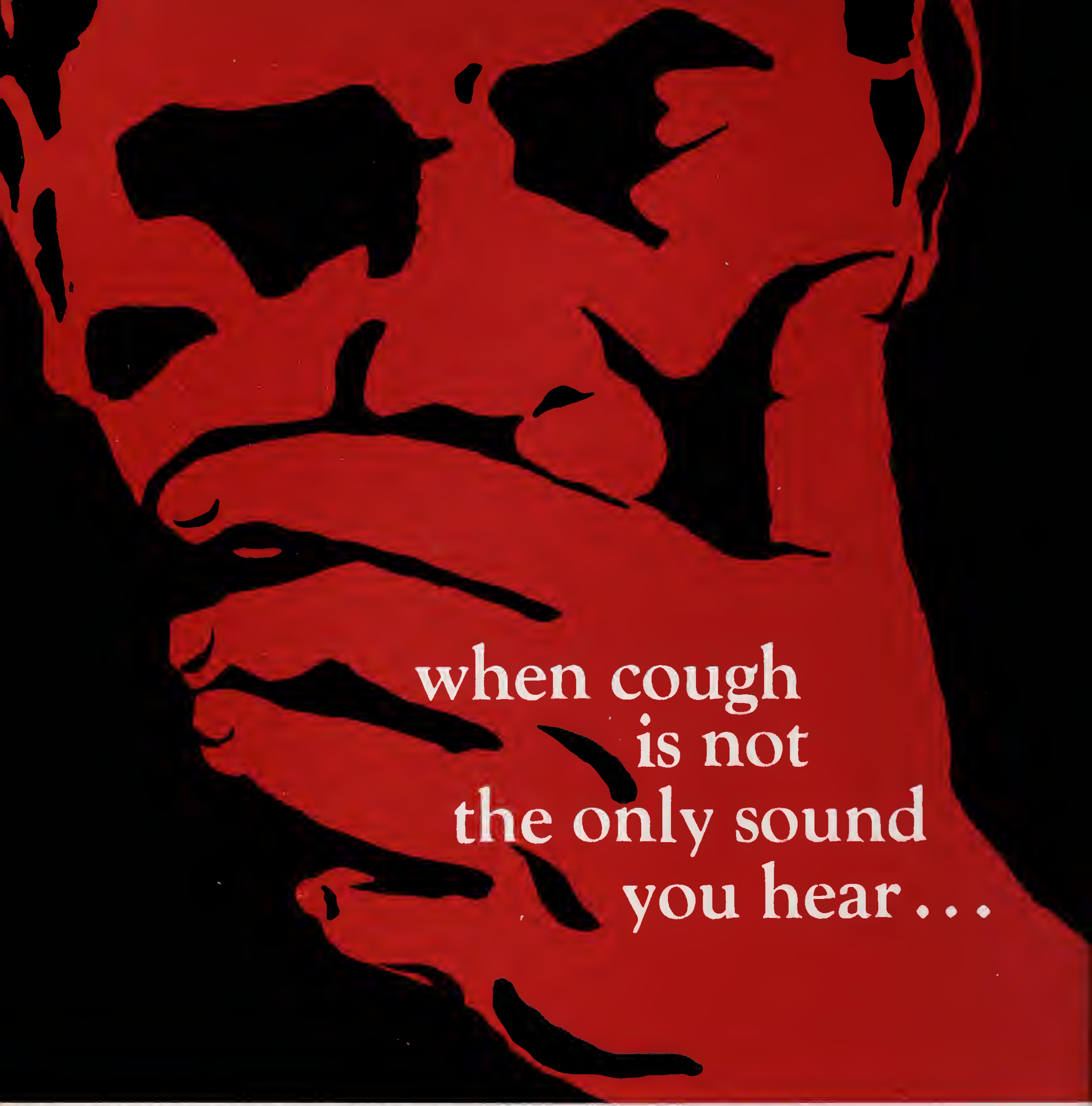

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Economical, efficient **b.i.d.** dosage—extremely well-tolerated by children, 6-12, and adults.

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Available on prescription only. Class X exempt narcotic. Permissible on oral prescription.

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Side Effects: Minimal, but when encountered may include jitteriness, nausea, drying of mouth, insomnia, constipation, which disappear upon adjustment of the dose or discontinuance of treatment.

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President's Page

JOIN DOCPAC FOR SURVIVAL

Thirty years ago the French system of compulsory health insurance began in earnest. It is government-sponsored, locally administered, and is based on occupational classification. Its coverage under Social Security operates by executive order and now embraces more than 22 million people. Fee schedules are imposed by the Social Security Commission which has no medical representation whatsoever. In 1960 it was decreed that certain fees would be paid for the services of doctors who subscribed to the plan, but less than half of these amounts would be returned to patients whose physicians refused to accept the schedule. Fees paid to certain categories of doctors were much smaller than those paid to so-called specialists for the same services. Finally, the doctors united and struck, but in spite of this the government achieved most of its goals, namely, to divide the profession, to set its fee schedules, and to control its activities. Medical practice had effectively been reduced to the level of a trade union.

But that was France. This is America. Can it happen here? Such changes are subtly gradual. The fee schedule for Medicaid was drafted by the D.C. Department of Public Health with the "advice" of a large committee, only a few of whose members are practicing physicians. The Civil Service Commission is openly aiming for complete medical coverage of all Federal personnel under its jurisdiction at controlled fees (a measure of coverage not found in any European health plan), and the cost of medical care is unjustly blamed upon the medical profession by most of the Nation's newspapers.

We as doctors can react to this growing control of medical practice in several ways. We can shrug it off as inevitable. We can become paid employees of the system and thus abrogate all vestiges of professional freedom. Or by political participation we can help to control the source of governmental domination.

The American Medical Association has established an organization called AMPAC to assist in the election of congressional candidates who favor independent medical practice. Most of the states have similar organizations. In the District of Columbia it is known as DOCPAC. With your help these organizations can become powerful. In 1966 CALPAC (California) by its intensive campaigning helped to elect a slate of 15 of 17 candidates to Congress who were favorable to private medicine. In Nebraska similar efforts almost single-handedly elected 2 Congressmen.

Most doctors dislike politics for professional gain, but it is time for us to become less aloof before it is too late. I urge you to join DOCPAC for your own survival.

William S McCune

Editorials

Metropolitan Washington Regional Medical Program in Operation

The first operational grant was awarded to the Metropolitan Washington Regional Medical Program, effective March 1, 1968, by the Division of Regional Medical Programs of the National Institutes of Health. The award for the first year, ending February 28, 1969, is $418,318, to support 3 initial projects as part of the Program's ultimate goal of bringing the latest advances in the diagnosis and treatment of heart disease, cancer, and stroke to the people in the Metropolitan Washington Region.

These projects are: A Hospital Stroke Station for the diagnosis, treatment, and investigation of cerebral vascular disease at Freedmen's Hospital; a Metropolitan Washington Region Cerebral Vascular Disease Follow-up and Surveillance System located at Georgetown University Medical Center; and a training program for cardiovascular technicians at the Washington Hospital Center for selected personnel from all Washington area hospitals.

The Freedmen's Hospital project establishes a team approach and long-term management of the stroke patient, which, as the study progresses, will provide a geographical separation of these patients to a stroke area for the acute management of the disease. It contains procedures for the evaluation of past and present treatment methods, procedures for the determination of the feasibility of management, including rehabilitation, on an outpatient status early in the course of the stroke, and, lastly, a prospective determination of the results of this type of improved care.

The Georgetown Medical Center project establishes a follow-up and surveillance system initially at Georgetown as a model for the Region. The services at both Georgetown and Freedmen's will ultimately be available to patients of all physicians in the Washington area.

The Washington Hospital Center project provides a program for training cardiovascular technicians for nursing and physician support and assistance in a highly technical, medical and surgical area in the Region. The scope of the training involves technical activities now performed in part by highly trained nurses, who are scarce, and other activities now performed in part by physicians, surgeons or engineering personnel. The program is of 2 years' duration, and while pilot in scope during the first year it subsequently will become truly Regional.

The Metropolitan Washington Region was one of the first 10 to become operational. Since the first operational grant was made, March 1, 1968, the Region has continued to develop new projects related to its basic plans. The Advisory Group of the Region at its meeting on May 1 reviewed and approved 10 projects which comprise a supplemental operational grant application totaling over $2,000,000 a year for the next 3 years. These include projects in areas of cancer, heart and stroke. Important in these operational projects are additional large programs for the stroke patients and projects for the establishment of mobile coronary care units.

In accepting the position as Coördinator for the Metropolitan Washington Regional Program in 1967 I did so with the understanding that I would leave the position once the planning was developed and the Program became operational. Because such progress has been made, as indicated above, I plan to leave the Program during the year. My best wishes for its continued success in the future.

Thomas W. Mattingly, M.D.
Coördinator, Metropolitan Washington Regional Program

Opinions expressed in contributions to the Editorial Section are those of the writers and do not necessarily reflect the views of the Medical Society of the District of Columbia

How Is Your Management?

The greatest void in medical education has always been, and still remains, the almost complete absence of a firm and comprehensive course of study dealing with "The Organization and Management of Medical Practice." In some instances the blame for this lack of education in this phase of medicine may be due to the lack of understanding and knowledge of the full-time teaching professors who have never actually been in private practice. These able professors are sheltered financially and physically by the university of their association and have no need for such knowledge. On the other hand, some professors who do private practice may say, "Let them (the students and interns) learn the way I did, by trial and error." Neither of these professors provide full and complete information on this subject to their students.

It is not enough in this modern and fast-moving world, with the vast media of communications, for the medical student to be taught the usual medical school curriculum alone without any knowledge and understanding of how to run an office, how to associate with nonmedical people-patients, how to select an office site, what type of office is best suited for his particular work, and all the other facets which help to make a better and more efficient physician, no matter whether he be general physician or specialist. Where does the young physician, who has been in school since the age of 6 until past his internship, and more than likely has never been thrust into the business world, gather this knowledge? Certainly not in our medical schools!

In view of this lack of knowledge the Committee on Medical Economics of the American Academy of General Practice decided the time is now to do something to fill this void for future physicians and particularly the future family doctor. This Committee, with the aid of members of the Society of Professional Business Consultants, a Chicago-based national association, prepared a 105-page manual titled "Organization and Management of Family Practice."

This is the first time any attempt has been made by any group in medicine to provide expert advice in this field. This volume comprises 14 sections and discusses the Specialty of Family Medicine, Functions of Family Physicians, Types of Practice, Selecting a Location, Office Layout, Equipping an Office, Office Assistants, Office Procedures, Office Policy, Diagnostic Procedures, Insurance for Your Practice, Personal Financial Planning, and 2 final sections dealing with Community Resources and Professional Associations.

As you can anticipate by the subject of each section, the manual is full of helpful facts and aids. It is free upon request to all medical students, interns, residents, and medical school and hospital libraries. Others may obtain a copy for $1.00 from the American Academy of General Practice, Volker Boulevard at Brookside, Kansas City, Mo. 64112.

For the specialist or family doctor, young or "old-timer" in practice there are many helpful suggestions on better methods of office procedure or on how to improve your present methods.

I. P. F.

Let's Prevent a Long Hot Summer

Speaking to the new President's Council on Physical Fitness and Sports, of which he is Chairman, Vice President Humphrey reported that:

- 1 of every 2 American adults is overweight, according to estimates by the American Medical Association;
- 50 per cent of America's college students—young men and women in their late teens or early twenties—fail to meet accepted physical fitness standards; and
- The average American youngster spends 10 hours watching TV for every hour he spends in supervised sports or physical activity.

As a way of getting Americans of all ages to be more active and fit, the Vice President has suggested that we convert our schools into neighborhood recreation centers. Specifically, he is asking all communities to keep open their school sports facilities—the gymnasiums, swimming pools, tennis courts, tracks, and playing fields—this summer.

Mr. Humphrey's request makes excellent sense. According to the Council, America has nearly $100 billion invested in its schools, yet most communities operate them at capacity only 8 hours a day, 180 days a year.

Communities which already employ the approach recommended by the Vice President report that it costs only a few dollars per capita to cover the cost of staff, equipment, maintenance, and other operating expenses. That's cheap compared to the cost of duplicating the trained leadership and extensive facilities which our schools employ only part-time.

Physicians more than any other group should realize the importance of physical fitness. We have an excellent opportunity to propagate the Council's message: Stimulate parents to work through Parent-Teacher Associations and school Boards of Education to keep the sports facilities open this summer. Any of you who are school physicians should impress this need on your school principals. This could bridge that gap to prevent this from being a LONG HOT SUMMER.

Margaret M. Kenrick, M.D.
Chairman, Committee on Rehabilitation

Physicians' Assistants—A New Health Career

An experimental curriculum designed to develop a new type of health professional—a college-trained personal assistant to the doctor able to relieve him of many of the routine diagnostic and therapeutic functions—is being initiated at Alderson-Broaddus College in Philippi, West Virginia, with a grant from the Commonwealth Fund. The Fund is providing $97,100 to the College over a 2-year period to support the planning and initial implementation of an experimental curriculum that will qualify students as Physicians' Assistants.

A liberal-arts institution with a well established tradition of combining general education with collegiate-level professional training, Alderson-Broaddus College will develop and conduct the Physicians' Assistant curriculum in association with the Broaddus Hospital and the Myers Clinic, both located in the town of Philippi, the West Virginia University Medical Center, in nearby Morgantown, and with the West Virginia State Medical Association, with headquarters in Charleston, which will provide advice and guidance to the experiment. All of these associated medical organizations are represented on a curriculum advisory committee established by the College to help its president and the faculty design and implement the experiment.

The objective of the curriculum is to prepare graduates to serve as professional associates to doctors by assuming responsibility for the more routine aspects of medical practice now customarily performed by the doctors themselves. With partial support from the Commonwealth Fund, a similar experiment is under way at Duke University School of Medicine for young men with some previous experience in the health field, usually in the medical corps of one of the military services. The Alderson-Broaddus curriculum would be addressed to high-school students, would include women as well as men, and would be the first at the baccalaureate level.

As presently conceived, the experimental curriculum would have 3 successive parts:

1. Liberal-arts and basic-sciences education, designed to provide the student with continued capacity for growth in his later professional career.
2. Clinical procedure and technology, to provide competence in the principles of the management of patients and diagnostic equipment and testing—electrocardiograms, urinalysis, and blood counts, for example, as well as the fundamentals of X-ray diagnosis.
3. Clinical clerkship training, to equip students with an understanding of the disease process, chronic and acute, and the doctor's decisions concerning them; during this period the student would learn how to take histories of patients, conduct physical examinations, and detect abnormal signs and symptoms.

The extensive clinical training involved in the proposed curriculum is made possible by the fact that the College operates on a 4-term, year-round calendar that includes periods which students devote to off-campus experiences related to their education.

The College expects to admit its first class, an anticipated 25 students, to the new 4-year degree course in September.

The West Virginia State Medical Association will participate in assessing the content and structure of the curriculum, with a view toward developing recommendations for licensure of graduates under the state's medical-practice laws, including qualifying examinations.

Statement on the Use of Normal (Whole, Pooled) Human Plasma

A COMMITTEE of the National Research Council has recommended that the use of whole, pooled human plasma "be discouraged and even discontinued" because of strong new evidence that it often transmits hepatitis.

The NRC Committee on Plasma and Plasma Substitutes based its recommendations on the report of a 3-year study by 2 Los Angeles researchers in which 10 per cent of the patients receiving the plasma became ill with acute hepatitis within 6 months. In all cases the plasma had been subjected to a commonly accepted 2-part sterilization procedure, consisting of storage in the liquid state at slightly greater than room temperature (86–90°F) for 6 months and then treatment with ultraviolet radiation.

Both the Committee's recommendation, in the form of a 1600-word statement, and the research report[1] appeared in the March–April issue of the journal *Transfusion*.

The study was conducted by Allan G. Redeker and Carl E. Hopkins, both of the University of Southern California School of Medicine and the Los Angeles County Hospital. Theirs is the first large-scale, prospective investigation of this problem to be undertaken.

Because plasma, the cell-free liquid portion of uncoagulated blood, does not deteriorate in the manner of whole blood it can be separated from outdated whole blood and preserved. Pooling, the mixing together of large quantities of plasma from the blood of many individuals, is widely practiced both as an economy and because it is thought that such mixing will neutralize dangerous antibodies. Pooling has been done commercially since the early 1950's; large quantities have also been produced locally by hospital and other blood banks since that time.

The Committee estimates that more than 300,000 units of pooled plasma are administered in the United States each year, primarily in the treatment of severely burned patients and as a plasma volume-expander for the severely injured. It is sometimes given as a nutritional supplement and in the treatment of coagulation and immune deficiencies, but there is little hard evidence to support these applications, the Committee notes.

Whole plasma has had a controversial history since its introduction in World War II, when it was employed to revive and sustain the badly wounded. It was used extensively during the War, and subsequently hundreds of thousands of surplus units were distributed and used throughout the United States.

Later it was shown that the use of plasma from large pools carried an unacceptable risk of transmitting hepatitis, presumably because of the presence of a virus. A major program of research failed to provide a practical solution until a report in the late 1940's suggested that prolonged storage of whole, pooled plasma at room temperature was virucidal. Further studies appeared to confirm this, and in 1958 the NRC Subcommittee on Plasma stated, on the basis of data then available, "that the storage of liquid plasma under these conditions (6 months at approximately 32°C) can be used to produce a product of minimal infectivity."

Now, however, Dr. Redeker's study has cast serious doubts on those assumptions. Using chemical and histologic methods not available to the earlier investigators, his controlled, prospective study, in contrast to the earlier retrospective studies, found that 12 of the 120 patients studied at Los Angeles County Hospital contracted acute hepatitis. Not a single case of hepatitis was noted among the 46 patients receiving the control solution, 5 per cent serum albumin.

"It is hoped," said the Committee on Plasma and Plasma Substitutes in its statement in *Transfusion*, "that this report of a soundly based and carefully conducted scientific investigation will be seriously received. To refute findings cited in this single unconfirmed study will require data derived from studies in the laboratory and clinic, not from controversial discourse. Because of the sizable resources, effort, and time required for such studies, however, the possibility that Redeker's work will be repeated in other laboratories appears slight. Even if other studies were initiated now, years would pass before requisite data would be forthcoming. In the interim Redeker's data appear convincing: combined treatment with ultraviolet irradiation and storage at 30–32°C for 6 months under closely controlled commercial supervision does not eliminate the risk of hepatitis."

"Accordingly," it concluded, "the Committee recommends that the use of whole, pooled human plasma be discouraged and even discontinued unless a clear-cut case can be made for its unique requirements."

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[1] REDEKER, A. G., HOPKINS, C. E., JACKSON, B., AND PECK, P.: A Controlled study of the safety of pooled plasma stored in the liquid state at 30–32 degrees C for 6 months. Transfusion, March–April, 1968.

Open-eyed nights

Too tense to sleep...too tired to get up. Early to bed, late to rise, and not much sleep at that, the patient with severe psychic tension is understandably tired. His tensions and overreactions to the day's stresses may interfere with proper sleep, and his inability to face the day's activities can produce an ever-worsening pattern. By relieving psychic tension, Valium® (diazepam) facilitates sleep, particularly with an h.s. dose. In many patients, the usefulness of Valium has been demonstrated in relieving psychic tension alone or with secondary depressive symptoms. Valium is generally well tolerated and, with proper maintenance dosage, usually does not unduly impair mental acuity or ability.

Before prescribing, please consult complete product information, a summary of which follows:

Indications: Tension and anxiety states; somatic complaints which are concomitants of emotional factors; psychoneurotic states manifested by tension, anxiety, apprehension, fatigue, depressive symptoms or agitation; acute agitation, tremor, delirium tremens and hallucinosis due to acute alcohol withdrawal; adjunctively in: skeletal muscle spasm due to reflex spasm to local pathology, spasticity caused by upper motor neuron disorders; athetosis, stiff-man syndrome, convulsive disorders (not for sole therapy).

Contraindications: Known hypersensitivity to drug; children under 6 months of age; acute narrow angle glaucoma; may be used in patients with open angle glaucoma who are receiving appropriate therapy.

Warnings: Not of value in treatment of psychotic patients, and should not be employed in lieu of appropriate treatment. As with most CNS-acting drugs, caution patients against hazardous occupations requiring complete mental alertness (*e.g.,* operating machinery, driving). When used adjunctively in convulsive disorders, possibility of increase in frequency and/or severity of grand mal seizures may require increase in dosage of standard anticonvulsant medication; abrupt withdrawal in such cases may also be associated with temporary increase in frequency and/or severity of seizures. Advise patients against simultaneous ingestion of alcohol and other CNS depressants. Withdrawal symptoms (similar to those with barbiturates and alcohol) have occurred following abrupt discontinuance. Keep addiction-prone individuals (such as drug addicts or alcoholics) under careful surveillance because of their predisposition to habituation and dependence. Use of any drug in pregnancy, lactation or in women of childbearing age requires that potential benefit be weighed against possible hazard.

Precautions: If combined with other psychotropics or anticonvulsants, carefully consider individual pharmacologic effects —particularly with known compounds which may potentiate action of Valium (diazepam), such as phenothiazines, narcotics, barbiturates, MAO inhibitors and other antidepressants. Employ usual precautions in the severely depressed or in those with latent depression; suicidal tendencies may be present and protective measures necessary. Observe usual precautions in impaired renal or hepatic function. Limit dosage to smallest effective amount in elderly and debilitated to preclude ataxia or oversedation (initially 2 to 2½ mg once or twice daily, increasing gradually as needed or tolerated).

Adverse Reactions: Side effects most commonly reported: drowsiness, fatigue and ataxia. Infrequently encountered: confusion, constipation, depression, diplopia, dysarthria, headache, hypotension, incontinence, jaundice, changes in libido, nausea, changes in salivation, skin rash, slurred speech, tremor, urinary retention, vertigo and blurred vision. Paradoxical reactions such as acute hyperexcited states, anxiety, hallucinations, increased muscle spasticity, insomnia, rage, sleep disturbances and stimulation have been reported; should these occur, use of the drug should be discontinued. Because of isolated reports of neutropenia and jaundice, periodic blood counts and liver function tests are advisable during long-term therapy. Minor changes in EEG patterns (low-voltage fast activity) observed during and after therapy and are of no known significance.

Dosage: Individualize for maximum beneficial effect. *Adults:* Tension, anxiety and psychoneurotic states, 2 to 10 mg b.i.d. to q.i.d.; alcoholism, 10 mg t.i.d. or q.i.d. in first 24 hours, then 5 mg t.i.d. or q.i.d. as needed; adjunctively in skeletal muscle spasm, 2 to 10 mg t.i.d. or q.i.d.; adjunctively in convulsive disorders, 2 to 10 mg b.i.d to q.i.d. *Geriatric or debilitated patients:* 2 to 2½ mg, 1 or 2 times daily initially, increasing as needed and tolerated. (See Precautions.) *Children:* 1 to 2½ mg t.i.d. or q.i.d. initially, increasing as needed and tolerated (not for use under 6 months).

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Drug Dependence: Where to Get Help*

ALTHOUGH SOCIETY doesn't permit people who are drug-dependent to have their own way, it does try to help them overcome their habit.

Many want help and seek it out; others have to be persuaded.

It is not easy for the average person to recognize someone who is abusing drugs. Symptoms are varied, sometimes vague, and often similar to those of other conditions. Drug abuse may be suspected, however, if the following signs are present:

— Inability to coördinate when standing or walking, muddled speech, and impaired judgment can indicate barbiturate abuse. This intoxication closely resembles drunken behavior, except that the individual doesn't smell of alcohol.

— Rapid pulse, restlessness, jittery muscular twitches, heavy sweating, and bad breath are hallmarks of amphetamine abuse. The individual also tends to be nervous, highly talkative, and overactive.

— Marihuana abuse typically leads to increased appetite with an especial craving for sweets. The user is often apathetic, listless, and careless about his personal habits.

— If a person has widely dilated pupils and constantly wears dark glasses, even at night, LSD or marihuana may be involved.

— Pinpoint pupils, on the other hand, are a sign of possible abuse of heroin or another narcotic drug. So are chills and needle marks on the arms and legs. Addicts often wear long-sleeve sweaters, even in summer, both to keep warm and to hide their scars.

Another way to tell if a person is taking drugs is to listen to the language he uses. The jargon of drug abusers has spilled over, to a certain extent, to the speech of the younger generation. "Turn on," "kicks" and similar expressions, once the property of the abuser world, are now in common usage. Certain words and phrases, however, are exclusively in the abuser's vernacular.

"Mainlining," for example, means injecting a drug into the veins.

"Pot head" and "acid head" refer respectively to abusers of marihuana and LSD.

"Barbs," "goofballs" and "candy" are popular slang for barbiturates.

Amphetamine abusers may refer to their drugs as "bennies," "co-pilots," or "wake-ups."

If you know someone who is dependent on drugs, the best thing you can do is get medical attention for him.

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* From "The Crutch that Cripples: Drug Dependence," a publication of the Committee on Alcoholism and Drug Dependence, Council on Mental Health, American Medical Association.

DR. WILLIAM S. McCUNE, President of the Medical Society of the District of Columbia, has just distributed a $25,000 grant from the American Medical Association Education and Research Foundation to representatives of Washington's 3 medical schools (left to right): Dr. Angus Griffin, Associate Dean of George Washington University ($9,270.85); Dr. McCune; Dr. John C. Rose, Dean of Georgetown University ($12,328.85); and Dr. K. Albert Harden, Dean of Howard University ($3,418.00). The funds are distributed according to each school alumni's contribution to AMA-ERF.

If you don't know a physician, call the medical society of the county you live in or a local hospital. They can give you the information you need.

Proper medical care is an essential first step. The physician will know whether to administer another drug to counteract the effects of the drug being abused. He will know how to treat other illness brought about by drug abuse. He will arrange for hospitalization if necessary.

He also will undertake the next step, often in consultation with a psychiatrist or with counseling services in the community. That step is the treatment of the mental and emotional disorders which preceded or resulted from drug dependence. As in all illnesses, the chances for recovery are better the earlier treatment is started.

HEALTH DEPARTMENT NOTES

Detoxification Center

ALCOHOLISM is a major public health problem in the District of Columbia as it is in most other large cities. It is estimated there are over 47,000 people in Washington who have drinking problems. Some 5,000 of these are chronic alcoholics.

The D. C. Department of Public Health is providing health care for alcoholics at the Rehabilitation Center for Alcoholics, located at Occoquan, Virginia. In operation for almost 2 years, the Center has admitted approximately 2,000 patients for diagnosis and treatment. Twenty-five per cent of the first admissions who were discharged have not returned. Data at the Center reveal that 128 patients who were treated are now sober, gainfully employed, and many have been reunited with their families. Surveys indicate that the majority of the patients feel they have been helped during their treatment period.

A second significant expansion of the program is the establishment of a Detoxification Unit which offers emergency care for alcoholics in lieu of incarceration. This service began during December of last year, and temporary quarters were made available at D. C. General Hospital where 700 persons were admitted to the Detoxification Unit.

In May 1968 the Health Department moved the Detoxification Center to a more permanent site at 619 N Street, N.W.

This 50-bed, short-term (3 or 4 days) nursing care facility is for acutely intoxicated persons having no serious medical or psychiatric complications. We anticipate a yearly capacity of 4,500 or more patients. The building has been especially designed for this purpose and is located in one of the highest drunk arrest areas in the District of Columbia.

Through the Center we are trying to find satisfactory alternatives for the unproductive and burdensome methods commonly used to handle drunk cases and to shift public intoxication problems from a criminal to a health jurisdiction.

Only consenting inebriates are brought to the Center by policemen, and at the present time only a limited geographical area is being served. The Center provides 24-hour service for patients. Before admission each patient is screened carefully by a nurse. Patients requiring intensive medical care are promptly transferred to a hospital. Others receive an examination, medication, special diet, counseling, encouragement to become involved in follow-up treatment, and assistance in meeting immediate needs for food, housing and clothing.

In short, this Detoxification Center provides nursing care and medical treatment with referral to health, welfare and rehabilitation follow-up services as a substitute for confinement, conviction, and punishment for public intoxication.

Although the Center has been in operation only a very short time we believe that the program is offering the following new and useful services to the alcoholic and the community:

1. Provides relief to the police, courts and correctional facilities by providing emergency care and assistance to individuals having a problem for which there is presently no effective solution within the criminal system.
2. Provides direct medical care for acutely ill patients.
3. Provides a positive psychological setting to encourage inebriates to do something about their drinking problem.
4. Makes available a resource to help end a drinking spree with medical support. Otherwise, the inebriate is prompted to continue his drinking in order to avoid alcohol withdrawal symptoms.
5. Offers an opportunity to detect and refer for early treatment other medical problems commonly found among alcoholics, thus avoiding many costly emergencies or chronic illnesses later.

MURRAY GRANT, M.D., D.P.H.
D. C. Director of Public Health

Treatment of the Alcoholic in the U.S.S.R*

MIKE GORMAN†

Preventive and therapeutic approaches to alcoholism in the U.S.S.R. are both dynamic and singularly flexible. They challenge to their very roots many of the bromides about the supposedly doctrinaire, quasi-theological nature of Soviet psychiatry.

We were prepared to expect—on the basis of earlier reports in the literature and a natural American suspicion of foreign behavior—a rather vehement denial of the existence of severe mental illness in the Soviet Union. We were almost hoping, after several weeks of our tour, for the long anticipated contention that a socialist society provided the ingredients for an end to the tensions and conflicts which cursed the family of man in other parts of the world. Our hopes were never realized; the frank discussions about alcoholism serve as a superb illustration of why they were not.

Without exception, all of the psychiatrists, educators and public health officers to whom we talked agreed that alcoholism was a most serious problem in the U.S.S.R. They referred to the Russian "alcoholic tradition," noting rather sadly that many special holidays in the year were always celebrated by excessive drinking among both the old and the young. To combat these harmful tendencies they have mounted a continuing and intensive educational campaign. Posters pointing up the evils of alcohol were seen in all the factories, schools and apartment houses we visited. A wide variety of anti-alcoholic films is shown at labor union, club and cultural association meetings, and narcologists lecture to these groups quite frequently.

Alcoholics compose a large part of the patient load in several of the psychoneurological dispensaries we visited; as high as 25 per cent of the total number of patients in the active treatment register of the dispensaries were alcoholics. The psychiatrists at the dispensaries, assisted by Social Service nurses, make a very determined effort to check out all of the environmental influences which contribute to the drinking of the patient; if he is a worker in a factory, they talk to the labor union and pinpoint those in the union who encourage him to drink. They also search out relatives and friends with whom he has been drinking, and they charge them with the responsibility of helping to change the patient's attitudes. Their therapeutic approach to alcoholism is dynamic, aggressive, and highly directive. They place the heaviest emphasis upon individual and group conditioning behavior. They do not—and this surprised us—place much stress upon the physiologic bases of alcoholism. This position is most clearly expressed by I. I. Loukomski:

"Regardless of any position as to the 'biological' basis of alcoholism, none of us doubt that the patient's own behavior plays an overwhelming part in the development of alcoholism and in the aggravation of the clinical picture of the disease."

Initial conditioning is done in a group therapeutic setting in either the hospital or the dispensary. Patients are assigned to arduous daily duties; failures to perform these duties are punishable by a vote of one's fellow patients in consultation with staff physicians. Although there is no organization in the U.S.S.R. comparable to our Alcoholics Anonymous, the treatment group is frequently visited by former alcoholics who relate their own experiences and point out the possibility of maintaining sobriety. The staff frequently reads to the groups letters from former patients and their relatives; these are then discussed within the group.

Activity in the industrial workshop is a very important part of the restorative regime for alcoholics. Hard work for which they are paid teaches these alcoholics "new moral values." Work which involves large groups of muscles stimulates the central nervous system. Work assignments should be tailored to the individual needs of the patient, but according to Loukomski "they should not only impart to the patient a feeling of 'muscular joy' as I. Pavlov used to say, but also the moral satisfaction brought by accomplishment and the awareness that he is a useful member of society."

In the specific treatment situation, the Russians have been using aversive therapies to produce a conditioned negative reflex to alcoholism since the early 1930's. They are quite sophisticated in the pharmacologic therapy of alcoholism. They regard Antabuse® as too dangerous in its side-effects for most patients; they prefer nicotinic acid, which they contend both sensitizes the organism to alcohol and restores the vitamin metabolism of alcoholics.

In the actual treatment of the alcoholic state

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* This is a portion of an address before the 124th Annual Meeting of the American Psychiatric Association in Boston, May 16, 1968. The complete paper, entitled "Soviet Psychiatry and the Russian Citizen," will be published in the *International Journal of Psychiatry*.

† Executive Director, National Committee Against Mental Illness, 1028 Connecticut Avenue, N.W., Washington, D. C. 20036.

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they employ a wide variety of neuroleptics, and again the precision and sophistication of their use are impressive. For example, Stelazine® is utilized in handling both acute and chronic alcoholic hallucinoses. For the treatment of delirum tremens (known as "white fever" in Russia), a wide variety of tranquilizers is applied. For compulsive forms of alcoholism Diazyle® is used, and in other forms of alcoholism Librium® is administered. Despite extensive dispensing of pharmacologic agents, the Russian narcologists to whom we spoke generally rejected undue reliance upon them as "mechanistic." As a leading Russian narcologist put it in a recent article: "Thus far little has been accomplished in the treatment of alcoholics through the use of modern psychotropic drugs. We must probe deeper. . .psychotherapy must play in it (alcoholism) an important part."

Although we hear much in America lately to the effect that the alcoholic is a sick person and should not be arbitrarily jailed, actual practice falls severely short of this ideal. In Russia, where the rhetoric on this matter is quite limited, performance is infinitely superior.

If an alcoholic is picked up on the streets by the police in Russia, he is taken to the police station, but he is not thrown into a jail cell. There is a separate unit of the jail, usually consisting of 4 to 6 beds, which is called a sobering-up station; it is manned by a doctor and either a feldsher or nurse. The important point here is that the first contact of the alcoholic is with a doctor. If the alcoholic has a heart condition, for example, only a doctor can determine this and act accordingly. If the alcoholic has the "white fever," the doctor can determine this quickly and either hospitalize him or use a drug to help him. If he doesn't have to go to the hospital, the alcoholic is given a bed in the unit to sleep if off. The next morning he is driven to his home or place of work by a police officer. The factory and the family are informed of his detention as an alcoholic. Either the patient or the family must pay the costs of the previous night's lodging and medical care.

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Are You Listening?

A RECENT ARTICLE in *The Journal of the American Medical Association* stated that physicians were becoming increasingly apathetic in the areas of religion, civic commitment, and political effort. I certainly hope that this is not true, but, if so, that the trend can be reversed. We physicians are opinion-makers and have an opportunity to see many people every day. As a whole we are respected and our views are respected.

Unfortunately, there is still currently widespread indifference to political activity among physicians in the District of Columbia. Most physicians seem to have little recognition of the profound effects of the legislative activity on Capitol Hill. This is not because of the lack of educational efforts by organized medicine, from the AMA on down to our District of Columbia Medical Society; it is just that we often don't listen.

As physicians we have been almost shamefully unaware of the effects of political decisions on our lives and on our practices. We seem never to learn the importance of utilizing the political potential we possess by simply being physicians. The members of the House and Senate on Capitol Hill almost never hear from us except in expressions of violent opposition to some proposed bill that would directly affect us. Through DOCPAC, and then through AMPAC, each of us, by joining, can aid in determining the types of bills that are approved by Congress. The money spent through DOCPAC and AMPAC aid in electing and reëlecting friends of medicine to the Senate and the House. Some of the money is used for educational purposes. By doing so, especially with the young, we assist in the future of our country.

If you have not yet joined, send $20 for an individual membership or $30 for a family membership to DOCPAC, 825 New Hampshire Avenue, N.W., Washington, D. C. 20037.

WILLIAM H. COOPER, M.D.
Chairman, D. C. Political Action Committee

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News & Personals

Summary of Executive Board Actions

Proceedings of May 27 Meeting

Dinner for the Board members was served in the Exhibit Hall of the Medical Society. Dr. Edward W. Nicklas, Chairman, presided at the meeting, which was attended by Drs. William S. McCune, Frank S. Bacon, Gudrun Augustin-Rueckert, Paul R. Wilner, Robert J. Coffey, Henry D. Ecker, Raymond T. Holden, John J. Hughes, and Mr. Thomas Sadler, Secretary. Present by invitation was Dr. Margaret M. Kenrick, Chairman of the Committee on Rehabilitation. The meeting was called to order by Dr. Nicklas at 7:20 p.m.

The minutes of the April 22, 1968 meeting were approved as submitted.

Committee Meetings: The Secretary reported the following committees had met since the last meeting of the Board:

April	24	Interdisciplinary Committee on Drug Dependency
	25	Committee on Aviation Medicine
	25	Traffiic Committee
	26	Committee on Continuing Education
	29	Subcommittee on Occupational Health
May	1	Fee Review Committee
	3	Liaison Committee with the Clergy
	6	Committee on Continuing Education
	7	Committee on Medical Practice
	8	Committee on Medical Licensure
	8	Recruitment Subcommittee, Regional Medical Program
	8	Liaison Committee with the Allied Professions
	13	Physicians' Placement Committee
	14	Board of Credentials
	14	Committee on Blood Banks
	20	Committee on Emergency Medical Service
	21	Committee on Public Information and Education
	21	Grievance Committee
	21	Committee on Relative Value Study
	22	Liaison Committee with the Clergy
	23	Traffiic Committee
	23	Committee on Medical Practice
	23	Subcommittee on Occupational Health
	24	Committee on Continuing Education

Special Activities: The following special activities had been held since the last Board meeting: reception in honor of Dr. Dwight L. Wilbur, President-elect of the American Medical Association; Physician-Clergy Symposium; presentation of AMA/ERF checks by Dr. McCune to Deans of the 3 medical schools in D. C.; presentation of a check for $5,400 by Dr. Nicklas, in behalf of the Health Foundation, to Children's Hospital.

Board of Credentials: On recommendation of the Board of Credentials, 3 applicants were elected to membership, namely:

Active: HAIKAZ MARTIROS GRIGORIAN, M.D. Office: 901–23rd Street, N.W. Born in Abadan, Iran. Medical degree: George Washington University, 1959. Specialty: Psychiatry.

Associate: FARUK T. OZER, M.D. Office: 11125 Rockville Pike, Rockville, Md. Born in Yozgat, Turkey. Medical degree: University of Istanbul, 1950. Specialty: Internal Medicine.

Resident-Intern: STEVEN GEORGE COOPERMAN, M.D. Office: 2211 Jefferson Davis Highway, Arlington, Va. Born in New York City. Medical degree: Northwestern University, 1966. Specialty: General Practice.

Approval was given to the following changes in membership status:

Transfers

Active to Associate: Maryanna Bremer Dotson
Associate to Active: Pearl B. Holly

Resignation

Active: Ervin A. Gombos

The dues of Dr. Crenshaw D. Briggs were remitted because of illness.

Committee on Rehabilitation: Dr. Kenrick joined the meeting to present details supporting the request of the Committee on Rehabilitation for a planning grant. The new project, she said, to be successful must benefit the entire Metropolitan Area, not the District of Columbia alone. For this reason approval of other area societies should be obtained for implementation of such a grant. Dr. Kenrick also recommended that a permanent committee be established to assure full continuity of such a project.

The Board approved in principle the request for a planning grant as described in Dr. Kenrick's letter of April 22, 1968 and voted to refer the matter for further consideration to the Medical Council of the Washington Metropolitan Area.

Dr. Kenrick next discussed the contents of her letter of April 29, 1968 regarding the proposed subway system which, as described in the letter, would not be physically accessible to the handicapped. After some consideration of the matter a motion was made and carried that Dr. Kenrick's letter, with appropriate changes in phraseology to be made by President McCune, be sent to a list of designated officials and to all local newspapers.

Honorary Membership: Dr. McCune reported that Dr. Louis M. Rousselot, Deputy Assistant Secretary for Health and Medicine, Department of Defense, was very happy about having been awarded Honorary membership status.

History of the Medical Society: Dr. Nicklas stated that he had had a discussion with Dr. Wallace M. Yater and Mr. Theodore Wiprud regarding the possibility of preparing a third volume of the Medical Society's history. The proposal emanating from the discussion was that Mr. Wiprud would write the history without compensation, the Society providing him with a dictating machine and a secretary to transcribe the material.

The Board approved the writing of Part III of the Society's history by Mr. Wiprud, without compensation, but the Board would have to approve in advance the expenses in connection with printing the history.

Scientific Assembly Progress Report: Dr. McCune outlined for the Board the scientific program.

Dr. Bacon stated that, in his opinion, the Board should begin immediately planning for the 1969 and 1970 Assemblies. His recommendation was approved.

Luncheon Meetings with Dr. Grant: Dr. McCune summarized the highlights of his first 2 luncheon meetings with Dr. Murray M. Grant. He reported that Dr. Grant would like the endorsement of the Society in his proposal to subsidize every medical student in the Washington medical schools $1,000 per year, with funds to come from the Department of Public Health. It was the general opinion of the Board that should such subsidizing materialize, the funds should not come from the budget of Public Health but from the budget of the District of Columbia. It was agreed that this topic be presented in a letter from the President to the Committee on Liaison with the Medical Schools, requesting their guidance in this matter, and that the Deans of the 3 medical schools also be contacted.

Other items mentioned by Dr. McCune as having been discussed with Dr. Grant were the 1970 Public Health budget, optometrists and their desire for hospital privileges, the pending proposal for a 400-bed addition to Children's Hospital, and a study to be undertaken of hospital emergency rooms.

Recommended Changes in Healing Arts Practice Act: Considerable discussion was held regarding the letter of May 14 from Dr. Mitchell W. Spellman, Chairman of the Ad Hoc Committee on Medical Licensure, and the recommendations submitted by the Committee for revisions in the Healing Arts Practice Act. The Board voted to approve the proposed revisions of the Healing Arts Practice Act.

The Board agreed that the work of the Ad Hoc Committee was not over and it must continue functioning until finalization of efforts to implement revisions of the Healing Arts Practice Act, and that all possible support and endorsement of the proposed revisions should be obtained from the Medico-Chirurgical Society, the D. C. Dental Society, Dr. Grant and the D. C. Department of Public Health, and the Dean of each medical school.

Committee on Liaison with Allied Professions: A motion was made and carried that the following recommendation of Dr. William T. Gibb, Chairman of the Committee on Liaison with Allied Professions, be accepted: That a permanent subcommittee be established consisting of 3 registered nurses from the District of Columbia Nurses' Association and 3 members of the Liaison Committee to examine current problems arising between physicians and nurses in the District of Columbia.

Physician Rounds at "Resurrection City": To a questionnaire asking for physicians to volunteer to make daily rounds at "Resurrection City," Dr. McCune reported that to date 144 physicians had responded affirmatively.

Other Business

1. The following changes recommended by the Committee on Relative Value Study were ap-

proved: (a) the recommendations of the Section on Otolaryngology; (b) recommendations regarding psychiatry with the exception of the item on group psychotherapy; (c) the recommendations of the Economics Committee of the Section on Obstetrics and Gynecology of the Medical Society and the Economics Committee of the Washington Gynecological Society.

2. The Board approved the request of Dr. John F. Potter, Chairman of the Ad Hoc Committee on Colon and Rectal Cancer Study Committee, that reprints of the report of its findings, published in the April 1968 issue of MEDICAL ANNALS, be forwarded to members of the Medical Society.

3. Dr. Nicklas read a letter of appreciation from Mrs. George W. Cooley for the memorial service for Mr. Cooley, February 18, and for "the impressive tribute contained in the recent issue of the MEDICAL ANNALS."

There being no further business, the meeting adjourned at 10:15 p.m.

GENERAL NEWS

Highlight of the **Annual House Staff Dinner of the Washington Hospital Center,** held at the Hotel Statler-Hilton, May 17, was the presentation of Gold-headed Canes to "physicians who have given outstanding service to The Washington Hospital Center or to the 3 parent hospitals." Recipients this year were Dr. Garnet W. Ault, of the Senior Attending Staff, Department of Proctology; Dr. Henry L. Darner, of the Senior Attending Staff, Department of Gynecology; and Dr. Thomas W. Mattingly, of the Consulting Staff.

The Harry H. Kerr Essay Awards for the best papers written by members of the Hospital Center staff were presented on the same occasion. The first prize of $100 went to Dr. Csaba L. Magassy for his paper, "Jejunal Autografts for Replacement of Infected Arterial Prosthesis." Other prizes went to Dr. Thomas Goodridge, Dr. Guillermo Gil Montero, and Dr. Hamid Sahebjami.

Many special activities marked the close of the academic year at **George Washington University**. The **General Alumni Association Dinner** was held on May 17 at the Mayflower Hotel. At this dinner awards were presented in appreciation to those who have contributed to the University through service and leadership. Among those cited for service were 3 members of the Medical Society. Dr. Charles S. Coakley received the 1968 Alumni Service Award. Among recipients of the 25-year teaching service awards were Dr. Virginia P. Beelar, Assistant Clinical Professor of Medicine, and Dr. Myer H. Stolar, Associate Clinical Professor of Medicine.

Medical alumni of the University began their annual observances, May 23, with a postgraduate course in ophthalmology. At their banquet at the Shoreham Hotel, May 24, 2 physicians, Dr. Lorenz E. Zimmerman and Dr. John Leo Madden, re-

Statler Hilton

GOLD-HEADED CANE AWARD WINNERS at the Washington Hospital Center Staff Dinner, May 17, are (standing, left to right): Dr. Thomas W. Mattingly, Dr. Henry L. Darner, and Dr. Garnet W. Ault. At the podium presenting the Canes is Dr. Herbert H. Schoenfeld, Chairman of the Awards Committee. Seated are the Reverend Warren Mace (left) and Dr. Charles W. Ordman, President of the Medical Staff.

DR. CLAYTON B. ETHRIDGE (right), retiring Associate Dean of George Washington University School of Medicine, receives best wishes from Dean John Parks at commencement exercises, June 2.

ceived Achievement Awards. Dr. Madden, of the Class of 1937, directs the Department of Surgery at St. Clare's Hospital, New York, and is a member of the faculy of New York Medical College. Dr. Zimmerman, who received both his undergraduate and medical degrees from George Washington, is Clinical Professor of Ophthalmic Pathology. At the Armed Forces Institute of Pathology he is Chief of Division B-General and Special Pathology, Chief of the Ophthalmic Pathology Branch, and Registrar of the Registry of Ophthalmic Pathology.

Dr. Clayton B. Ethridge, who retired, June 30, as Associate Dean and Medical Director of George Washington Hospital, was presented with a University Chair.

At the dinner meeting Dr. William Brainin succeeded Dr. Jerome H. Epstein as Alumni President. In charge of the dinner arrangements was Dr. Robert F. Dyer.

Fifteen physician-parents participated in the George Washington University graduation exercises, June 2. Among them were 4 members of the D. C. Medical Society, Drs. Gordon R. MacDonald, John J. Francis, Ardeshir B. Irani, Jr., and Reginald S. Lourie, who presented medical diplomas to their sons, Drs. Gordon R. MacDonald, Jr., Jack D. Francis, Sands K. Irani, and Ira S. Lourie, respectively.

All physicians are invited to join a new organization being formed to support the Washington National Symphony. The group, to be called the **Physicians Committee for the Washington National Symphony,** will have fund-raising and public relations as its primary activities, and will also offer an opportunity for its members to share their enthusiasm for music and musicians. In addition to working for the orchestra, it is anticipated that members will be able to attend orchestra rehearsals, receptions, and other functions. Those interested in joining the Physicians Committee for the Washington National Symphony should contact Dr. Theodore R. Bledsoe, 10401 Old Georgetown Road, Bethesda, Md. 20014; telephone 469–9190.

The Department of Psychiatry of Georgetown University School of Medicine will offer a series of **postgraduate seminars** on the use of current psychiatric techniques by the medical practitioner. Meetings will be held weekly, on Thursday, for 12 weeks, beginning September 19, from 4 to 6 p.m., at Georgetown University Hospital.

The aim of the seminars will be to assist the clinician in identifying and managing various emotional difficulties in his patients. Discussions will focus primarily on the patients of the participating physicians and will be supplemented by patient interviews, discussion of current literature, and case presentations by the seminar staff. Topics

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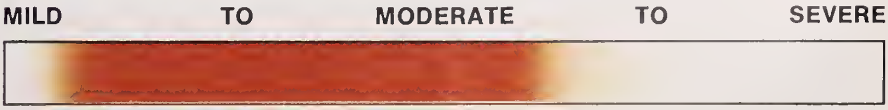
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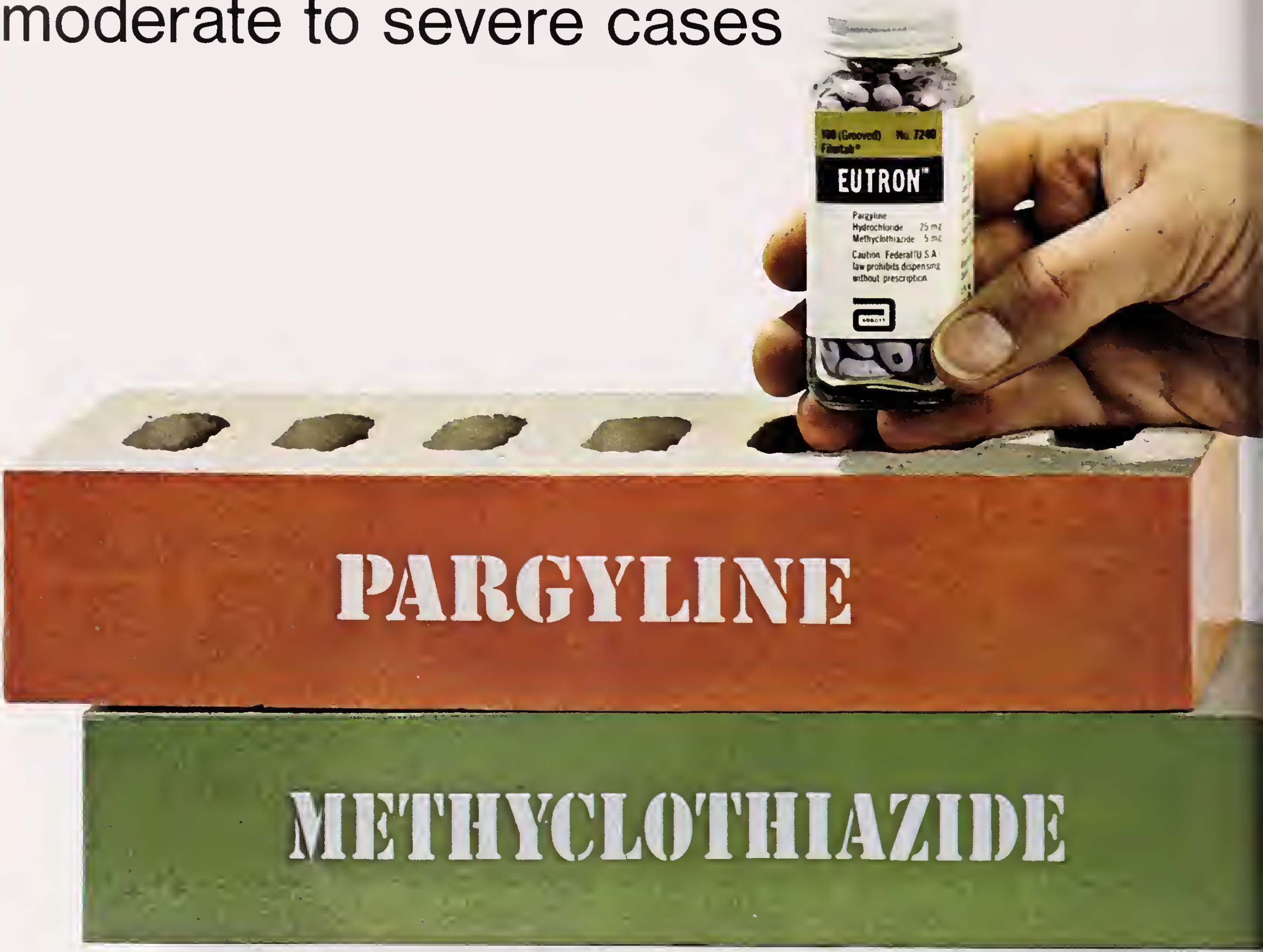
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Each tablet contains
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Indications: Edema and mild to moderate hypertension (Enduron), and mild to moderately severe hypertension (Enduronyl). More potent agents, if added, can be given at reduced dosage.

Contraindications: Sensitivity to thiazides; severe renal disease (except nephrosis) or shutdown; severe hepatic disease or impending hepatic coma (hepatic coma due to hypokalemia has been reported in patients on thiazides). Do not use Enduronyl in severe mental depression, suicidal tendencies, active peptic ulcer, or ulcerative colitis.

Warnings: Consider possible sensitivity where there is history of allergy or asthma. If added potassium is indicated, dietary supplementation is recommended. Reserve enteric-coated potassium tablets for cautious use only when necessary, as they may induce serious or fatal small bowel lesions (stenosis with or without ulceration), cause obstruction, hemorrhage, and perforation often requiring surgery; discontinue them immediately if abdominal pain, distention, nausea, vomiting, or g.i. bleeding occurs. Neither Enduron nor Enduronyl contains added potassium.

Precautions: Use thiazides cautiously in severe renal dysfunction, impaired hepatic function or progressive liver disease; also in pregnancy (bone marrow depression, thrombocytopenia, and altered carbohydrate metabolism have been reported in certain newborn). In surgery, thiazides may reduce response to vasopressors, and increase response to tubocurarine. Antihypertensive response may be enhanced following sympathectomy. Watch for electrolyte imbalance (e.g., hyponatremia) in all patients. In hypokalemia (especially in digitalized patients) give supplemental potassium. In hypochloremic alkalosis, give supplemental chloride.

Use rauwolfias with caution in patients with history of peptic ulcer. Rauwolfias with anesthetics may produce hypotension and bradycardia. Discontinue Enduronyl two weeks before elective surgery. Consider vagal blocking agents during emergency surgery. In epilepsy, adjust anticonvulsant dosage. In electroshock, shorten stimulus strength and duration. In occasional patients with depressive tendencies, rauwolfias may precipitate severe mental depression that usually disappears when drug is stopped.

Adverse Reactions: Thiazide reaction include blood dyscrasias (thrombocytopenia with purpura, agranulocytosis, aplastic anemia); elevation of BUN, serum uric acid or blood sugar; anorexia, nausea, vomiting, diarrhea, headache, dizziness, paresthesia, weakness, skin rash, photosensitivity, jaundice, symtomatic gout, and pancreatitis. Cutaneous vasculitis in the elderly has been reported with other thiazides. Adverse effects with deserpidine are qualitatively similar to those with reserpine, but their incidence is lower. These include nasal stuffiness, abdominal cramps or diarrhea, nausea, headache, weight gain, reduced libido and potency, peptic ulcer aggravation, epistaxis, skin eruption, asthma in susceptible patients, electrolyte imbalance, excessive salivation, and a reversible Parkinson's syndrome. Excessive drowsiness, fatigue, weakness, and nightmares may signal mental depression. Thrombocytopenia, purpura, and a symptom manifested by dull sensorium, deafness, uveitis, glaucoma, and optic atrophy are rare allergic reactions to other rauwolfias. Hypotension from antihypertensive agents may precipitate angina attacks in susceptible individuals. Usually adverse reactions disappear when drug is withdrawn.

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Each tablet contains
Pargyline Hydrochloride 25 mg.
with Methyclothiazide 5 mg.

Indications—Moderate to severe hypertension.

Contraindications—Pheochromocytoma, paranoid schizophrenia, hyperthyroidism and advanced renal failure. Not recommended in malignant hypertension, children under 12, pregnant patients.

Do not use with: centrally or peripherally acting sympathomimetic drugs; foods high in tyramine (e.g., aged and natural cheeses); parenteral reserpine or guanethidine; imipramine, amitriptyline, desipramine, nortriptyline or their analogues; other monoamine oxidase inhibitors; methyldopa or dopamine; separate Eutron and these agents by two weeks.

Sensitivity to thiazides; severe renal disease (except nephrosis) or shutdown; severe hepatic disease; impending hepatic coma from thiazide-induced hypokalemia.

Warnings—Patients: 1. No other drugs (particularly "cold preparations" and antihistamines), cheese or alcohol without physician's consent. 2. Promptly report orthostatic symptoms, severe headache, other unusual symptoms. 3. Angina pectoris or coronary artery disease patients must not increase physical activity with improved anginal symptoms or well-being.

Physicians: 1. Use antihistamines, hypnotics, sedatives, tranquilizers and narcotics (meperidine contraindicated) cautiously in reduced doses. 2. Stop Eutron two or more weeks before elective surgery; in emergency surgery reduce premedication (narcotics, sedatives, analgesics, etc.) to 1/4 to 1/5; carefully adjust anesthetic dosage to patient response. 3. Use cautiously in advanced renal failure. 4. Pargyline may induce hypoglycemia. 5. Consider possible sensitivity reactions when a history of allergy or asthma is present. 6. If potassium is indicated, dietary supplement is recommended; enteric-coated potassium tablets may induce serious or fatal small bowel lesions (stenosis with or without ulceration), cause obstruction, hemorrhage, and perforation frequently requiring surgery; discontinue medication immediately if abdominal pain, distention, nausea, vomiting or gastrointestinal bleeding occurs; Eutron does not contain added potassium. 7. Possible systemic lupus erythematosus has been reported for thiazides.

Precautions—Pargyline: Use cautiously at reduced dosage: caffeine, alcohol, antihistamines, barbiturates, chloral hydrate, other hypnotics, sedatives, tranquilizers, narcotics. Periodically do urinalyses, blood counts, liver function tests, etc. Use with caution in liver disease. Watch for orthostatic hypotension, especially in impaired circulation (e.g., angina pectoris, coronary artery disease, cerebral arteriosclerosis); also, augmented hypotension in concomitant febrile illnesses. Reduce or discontinue if hypotension is severe. In impaired renal function watch for cumulative drug effects, elevated BUN and other evidence of progressive renal failure; withdraw drug if these persist. In surgery increased central depressant response (hypotension and increased sedative effect) can be controlled by (1) discontinuing at least two weeks prior; (2) in emergency surgery lowering dose of premedication; (3) when necessary, administering a vasopressor. Do not use in hyperactive and hyperexcitable patients. Pargyline may unmask severe psychotic symptoms where emotional problems pre-exist. Use cautiously in Parkinsonism, especially with antiparkinsonian agents. In prolonged therapy, examine for change in color perception, visual fields, fundi and visual acuity. Also, prolonged therapy has made certain patients refractory to nerve blocking effects of local anesthetics.

Methyclothiazide: Use cautiously in severe renal dysfunction, impaired hepatic function or progressive liver disease; also in pregnancy (bone marrow depression, thrombocytopenia, and altered carbohydrate metabolism have been reported in certain newborn). In surgery thiazide may reduce vasopressor response and increase tubocurarine response. Antihypertensive response may be enhanced following sympathectomy. Watch for electrolyte imbalance (e.g., hyponatremia). Give supplemental chloride if hypochloremic alkalosis occurs and supplemental potassium if hypokalemia occurs (especially in digitalized patients). Thiazides may decrease serum P.B.I. without signs of thyroid disturbance.

Adverse Reactions — Pargyline: Orthostatic hypotension and associated symptoms, mild constipation, fluid retention, edema, dry mouth, sweating, increased appetite, arthralgia, nausea, vomiting, headache, insomnia, difficult in micturition, nightmares, impotence, delayed ejaculation, rash, purpura, weight gain, hyperexcitability, increased neuromuscular activity and other extrapyramidal symptoms. Drug fever is extremely rare. Reduction in blood sugar and hypoglycemic effects are possible. Congestive heart failure has been reported in a few patients with reduced cardiac reserve.

Methyclothiazide: Blood dyscrasias (thrombocytopenia with purpura, agranulocytosis, aplastic anemia); elevation of BUN, blood sugar or serum uric acid (gout may be induced); anorexia, nausea, vomiting, diarrhea, headache, dizziness, paresthesia, weakness, skin rash, photosensitivity, jaundice and pancreatitis. Cutaneous vasculitis in elderly patients has been reported with other thiazides.

If side effects are severe or persist, reduce dosage or withdraw drug. 804438R

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chosen will be of interest to internists, general practitioners, pediatricians, obstetricians, and others wishing to increase their skills in dealing with emotional problems.

Registration will be limited. Further information may be obtained from Dr. Louis F. Rittelmeyer, Jr., Department of Psychiatry, Georgetown University Hospital, Washington, D. C. 20007; telephone 625-7351.

Mr. James L. Harrison, 14th Public Printer of the United States Government Printing Office, is the new **President of the District of Columbia Division of the American Cancer Society.** He will succeed Dr. C. Roger Kurtz on September 1.

DR. ROBERT S. JASON (right), of Howard University College of Medicine, receives the St. George Medal and Citation for outstanding contributions to cancer control at the Cancer Society's annual meeting, May 8, from Dr. C. Roger Kurtz, who will retire as President, September 1.

At the meeting of the Board of Trustees following the Annual Meeting of the Society in the Medical Society's Exhibit Hall, May 8, Dr. S. Joseph Cosimano, Jr., Dr. LaSalle D. Leffall, Jr., and Mr. John F. Feeney were elected Vice Presidents. Among those elected to the Board of Trustees were Dr. William W. Funderburk and Dr. Nina B. Woodside.

Named to the Society's Executive Committee, in addition to the officers, were, among others, Drs. Kurtz, Robert S. Jason, Calvin T. Klopp, Cyril A. Schulman, and James G. Sites.

For 1968–69 the following officers will serve the **District of Columbia Thoracic Society:**

Dr. Maurice A. Sislen, *President;* Dr. Harold M Silver, *President-elect;* Dr. Roscoe C. Young, Jr., *Vice President;* and Dr. P. Gregg Rhodes, *Secretary-Treasurer.*

Dr. Moe Weiss was elected Representative Councilor to the American Thoracic Society, and Drs. Paul C. Adkins, Barrington B. Barnes, Robert F. Donohoe, and Colonel Forrest Pitts, MC, USA, were elected members-at-large.

These officers were chosen just prior to the joint annual meeting of the D. C. Tuberculosis and Respiratory Disease Association (the new official name of the Association) and the D. C. Thoracic Society. The announcement of the change in name was made at the annual meeting by President Richard W. Brown, who explained that the new name will better reflect the broader goals of the Association.

The Section on Radiology of the Medical Society of D. C. has elected Dr. Louis Winer as Chairman-elect for 1968–69. Dr. Winer will also serve as Secretary and Treasurer. Dr. Gregory T. Henesy is this year's Chairman.

The Washington Urological Society elected new officers at its April meeting. They are:

Dr. William P. Herbst III, *President-elect;* Dr. Arthur J. Wilets, *Secretary;* and Dr. G. Roland Gable, *Treasurer*

Dr. Robert E. Byrne took office as President for 1968–69.

Commissioner James L. Goddard, of the Food and Drug Administration, has resigned his position, effective July 1, to head the Atlanta, Ga., office of a new computer information organization, EDP Technology.

Dr. James E. Banta, former director of medical programs for the U. S. Peace Corps, has joined the faculty of the Georgetown University School of Medicine. Effective May 1, he became Director of the Division of Preventive Medicine and International Health. Since 1965 he has been Director of the Office of International Health of the Public Health Service. Dr. Banta is a graduate of Marquette University School of Medicine and holds a Master's degree in Public Health from Johns Hopkins University.

The position of **Distinguished Physician in the Veterans Administration** was recently established, and the **first appointee is Dr. William B. Castle,** Professor of Medicine at Harvard University. The post, as announced by Dr. H. Martin Engle, Chief Medical Director of VA, is designed to bring physicians to the VA "who have made very significant contributions to medical science and have obtained exceptional professional stature over long and distinguished careers." Dr. Castle has been associated for almost 50 years with the Harvard University Medical School and with hospitals in the Boston area. He will be stationed at the VA Hospital in West Roxbury, Mass., beginning in August.

Following the death of Dr. John M. Orem, President of Sibley Memorial Hospital, **Mr. Donald R. Ernst** has been appointed Acting President of the Hospital by the Executive Committee of the Board of Trustees. Mr. Ernst has been a member of the Board of Trustees for 18 years, serving as Treasurer of the Hospital and Chairman of the Investment and Finance Committee.

Mr. Russell F. Staudacher, Executive Director of the Student American Medical Association, submitted his resignation as of June 30 at the SAMA's 18th Annual Meeting in Detroit. Appointed Executive Director Emeritus, he will devote his time to fund-raising for the organization. **Mr. Robert Chapin,** Assistant Executive Director, was **appointed Acting Executive Director** for a 1-year period.

PERSONALS

The Achievement Award for 1968 of the Sibley Medical Association was presented to **Dr. Milton F. Weingarten** at the annual dinner-dance of the Association on March 30. Dr. Weingarten, along with Drs. James E. Boland, J. Phelps Hand, Richard F. Manegold, and the late Fuad Kaigni, were the founders of the Sibley Alumni Association, which is now the Sibley Medical Association.

Dr. James G. Sites, Chairman of the Department of Obstetrics and Gynecology at George Washington University, has been honored by the 1968 seniors, who dedicated their Yearbook to him, acknowledging "his qualities of leadership, understanding and compassion for the patient as a person." The 1964 seniors also dedicated their Yearbook to him.

Dr. Peter A. Santucci, Chief of Psychiatry at Holy Cross Hospital in Silver Spring and psychiatric consultant to Catholic University, was elected President of the National Guild of Catholic Psychiatrists at the Guild's 20th Annual Meeting in Boston in May. **Dr. John R. Cavanagh,** of Washington, was elected Secretary. The Guild will hold its next annual meeting in Washington in April, 1969.

DR. FREDERIC G. BURKE (right), Georgetown's Pediatrician of the Year, accepts a work of art as a reminder of the occasion and the citation from Dr. Philip L. Calcagno.

Dr. Frederic G. Burke was named Pediatrician of the Year by the Department of Pediatrics of Georgetown University School of Medicine. Dr. Burke, Professor of Pediatrics, was cited for "developing teaching programs in pediatrics, serving as a member of the first National Advisory Council to the Institute of Child Health and Human Development, and developing a model for intermediate hospital care at the Hospital for Sick Children in Washington, D. C." At a banquet, May 8, Dr. Philip L. Calcagno, Chairman of the Department of Pediatrics, presented Dr. Burke with a glass model of the Luxembourg Group, designed by Galt & Brothers of Washington.

Dr. David H. Kushner was cited for his services as Chairman of the Membership Committee of the International Fertility Association during the Sixth World Congress on Fertility and Sterility in Tel Aviv, Israel, May 20–28. At the meeting he presented a paper, in behalf of the staff of Columbia Hospital for Women, on "The Surgical and Obstetrical Complications of the IUD."

At a recent meeting in Boston of the American College of Physicians, the following members of the Medical Society were elected to fellowship: **Drs. Lewis H. Biben, Cyril A. Schulman, Jay R. Shapiro, and Stanley M. Silverberg. Dr. Morton C. Gluck** was elected an Associate member of the College.

Don't let monilia cut broad-spectrum therapy short...

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complex-nystatin

Use of broad-spectrum antibiotics can cause fungal overgrowth in the alimentary tract... and give rise to symptoms so troublesome that therapy must be prematurely stopped. Tetrex-F (tetracycline phosphate complex-nystatin) helps you circumvent this problem.

The nystatin can prevent overgrowth of monilia; the phosphate complex delivers tetracycline to the blood rapidly. Side effects are infrequent.

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When you start with economical Tetrex-F (tetracycline phosphate complex-nystatin), you can complete the full course of broad-spectrum therapy with less chance of losing control elsewhere. A good start for a healthy finish.

PRESCRIBING INFORMATION. For complete information consult Official Package Circular. *Indications:* Infections of respiratory, gastrointestinal and genitourinary tracts and skin and soft tissues due to tetracycline-sensitive organisms, in patients with increased susceptibility to monilial infections. *Contraindications:* The drug is contraindicated in patients hypersensitive to its components. *Warnings:* Photodynamic reactions have been produced by tetracyclines. Natural and artificial sunlight should be avoided during therapy. Stop treatment if skin discomfort occurs. With renal impairment, systemic accumulation and hepatotoxicity may occur. In this situation, lower doses should be used. Tooth staining and enamel hypoplasia may be induced during tooth development (last trimester of pregnancy, neonatal period and childhood). *Precautions:* Bacterial superinfections may occur. Infants may develop increased intracranial pressure with bulging fontanels. In gonorrheal therapy, serologic tests for syphilis should be conducted initially and monthly for 3 months. *Adverse Reactions:* Glossitis, stomatitis, nausea, diarrhea, flatulence, proctitis, vaginitis, dermatitis, and allergic reactions may occur. *Usual Adult Dosage:* 1 capsule q.i.d. Continue for 10 days in Beta-hemolytic streptococcal infections. Administer one hour before or two hours after meals. *Supplied:* Capsules, bottles of 16 and 100. Each capsule contains tetracycline phosphate complex equivalent to 250 mg. tetracycline HCl activity and 250,000 units of nystatin. For Oral Suspension, 125 mg. tetracycline and 125,000 u. nystatin/5 ml., 60 ml. bottles.

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CORRESPONDENCE

WASHINGTON, D. C., May 10, 1968

TO WARREN D. BRILL, M.D., CHAIRMAN,
COMMITTEE ON PUBLIC INFORMATION AND EDUCATION:

The following physicians served as judges at the 22nd Annual Science Fair held at the D. C. National Guard Armory on April 23, 1968.

Emile C. Nash
Orville W. Donnelly
John C. Bartone, Jr.
Frank N. Miller, Jr.
Nelson G. Goodman
Samuel B. Rosser
Ajaib S. Sidhu
Henry D. Bell
Warren J. Strudwick
Vincent Roux
Michael J. Halberstam

Dr. Lamberton and Mr. Steven Hopkins of the Department of Science of the D. C. Public Schools as well as the exhibitors seemed well pleased with the manner in which the judging of the Medical Science Section was conducted. Indeed many of the judges expressed great pleasure derived from many of the exhibits that they evaluated.

LEWIS H. KURTZ, M.D.
Chairman, Medical Science Judges

OBITUARIES

Henry Cook Macatee, M.D.

(1878-1968)

THE MEDICAL SOCIETY of the District of Columbia owes a deep debt of gratitude to Dr. Henry C. Macatee. He served the organization for many years as its Secretary, then its President, and finally as its delegate to the American Medical Association.

It was in the latter capacity I got to know him. And what a fine man he was—quiet, self-effacing, and gentle. If these qualities suggest an individual who never took a stand on a controversial issue it would in Dr. Macatee's case be misleading. For "Mac," as I called him, was a thoughtful man, and when the need arose he would act forcefully.

I shall never forget his prophetic and last talk before the AMA House of Delegates. Simply and eloquently he told the House that the Association's policies with regard to the delivery of medical care were not realistic and that it had to move along with the times. This, mind you, was in the 1940's when it took courage to speak to the House as Dr. Macatee did. I was very proud of him and told him so.

He refused to "run" for reëlection as a delegate, thinking his record spoke for itself. Regretfully, he was not reëlected. I felt then, and I do now, that Dr. Macatee's departure from the House was a great loss to American medicine.

Prior to World War II meetings of the Medical Society were held each week on Wednesday night. One meeting a month was devoted to business. The "old-timers" made it a point at these sessions to engage in parliamentary tactics in an effort to confuse and embarrass the President. It was a game they enjoyed immensely. If the proceedings became snarled too much Dr. Macatee took a hand. Quietly he straightened things out.

HENRY C. MACATEE, M.D.

I said to him several years later that he was the best parliamentarian that I had ever known. He replied that I was too generous but that he "always knew where the rabbits were."

The world is much better for having had a man like "Mac." And I am certain that I shall never see his like again.

THEODORE WIPRUD

Dr. Henry C. Macatee, one of Washington's most distinguished physicians, died, May 23, 1968 at the Washington Hospital Center. His long record of community service and medical practice comprised a span of 63 years, beginning in 1901.

Dr. Macatee was born in Front Royal, Virginia, August 4, 1878, to Charles Augustus and Mary Cook Macatee. After attending the public schools of Front Royal and Randolph-Macon Academy he enrolled for premedical study at Columbian College and received his medical degree from Columbian (now George Washington) University in 1900. He interned at Garfield Memorial Hospital the following year and thus began his long association with this hospital, which later merged into the Washington Hospital Center.

Dr. Macatee, a specialist in internal medicine, was on the attending staff of Emergency Hospital from 1904 to 1918; he was associate attending physician at Garfield Hospital from 1924 to 1937 and consulting physician from 1937. He was President of the Medical Staff of Garfield from 1935 to 1944.

He was long on the teaching staff of George Washington University, first as Instructor in Medicine, 1905 to 1910; later Professor of Therapeutics, 1923 to 1929; and Clinical Professor of Pediatrics from 1929 to 1937.

He served in the United States Naval Reserve from 1913 to 1919 and went on active duty during World War I. He was a medical advisor to the Draft Board, 1917–1918, and served as a member of the Appeal Board of Selective Service during World War II.

In community service Dr. Macatee's interests and activities were numerous. He was Medical Inspector of the Public Schools from 1905 to 1920; President of the Central Admitting Bureau for Hospitals from 1936 to 1938, when it merged with Health Security Administration, and President of the latter from 1938 to 1948. He served on the Board of the Community Chest and was active in the Kalorama Citizens' Association.

Dr. Macatee became a member of both the Medical Society of the District of Columbia and the Medical Association of the District of Columbia, which later merged with the Society, in 1902. He was Recording Secretary of the Medical Society from 1905 to 1920, President in 1921, and delegate to the American Medical Association from 1929 to 1943. In 1943 he became a Life member of the Society. Dr. Macatee served the Medical Society in many other ways through committee work and as the Society's representative.

He was a member of the American Medical Association, a Fellow of the American College of Physicians, a member of the Washington Academy of Sciences, the George Washington University Medical Alumni Association, the Hippocrates-Galen Medical Society, and an honorary member of the Clinico-Pathological Society. He was a longtime member of the Cosmos Club, the Chevy Chase Country Club, and a member and former elder of the Presbyterian Church of the Pilgrims.

Surviving him are 2 daughters, Mrs. Mary M. Donaldson, of 4009 Underwood Street, and Mrs. Gertrude M. Butler, of 3708 Spring Street, both of Chevy Chase, Md.; a sister, Mrs. Charles B. Calvert, of Washington; and 2 brothers, Robert B. Macatee, of Washington, and Colonel Edward V. Macatee, U.S.A. (Ret.), of Front Royal, Va.

Dr. Macatee was associated in practice with the late Dr. J. Russell Verbrycke, Jr., for many years. For the December 1949 issue of the MEDICAL ANNALS, Dr. Verbrycke contributed a detailed account of Dr. Macatee's life and accomplishments and a warm appraisal of the unique character of his friend.

Walter R. Gladding, M.D.

(1898–1968)

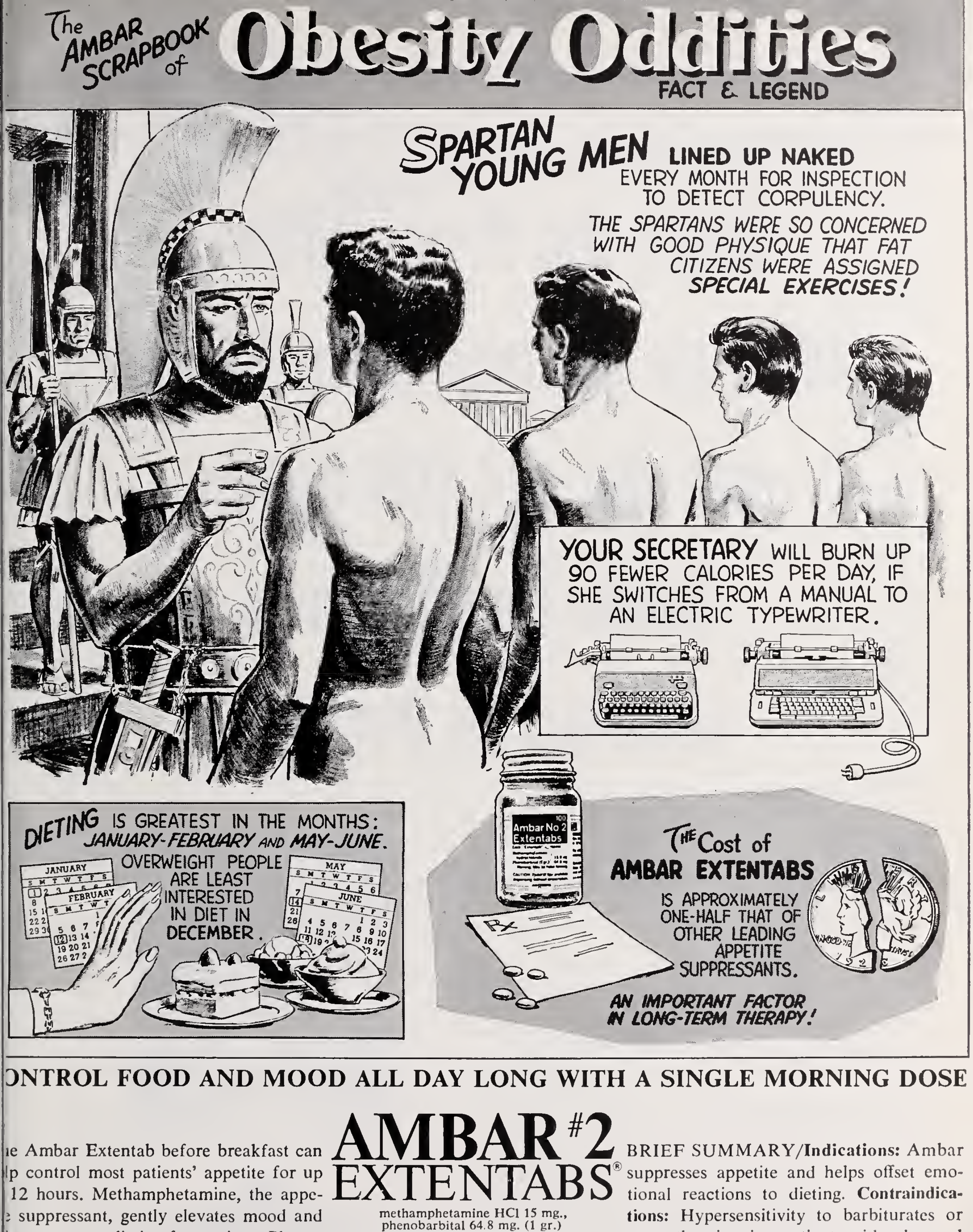
DR. WALTER R. GLADDING, a former member of the Board of Veterans Appeals, died, May 26, 1968 at the Veterans Center in Martinsburg, West Virginia. He lived at 7407 Meadow Lane, Chevy Chase, Md.

Dr. Gladding was born in New Church, Va., June 28, 1898. After premedical study at the University of Richmond and William and Mary College, in Williamsburg, he enrolled at the Medical College of Virginia in Richmond, receiving his medical degree in 1928. He interned at Sheltering Arms Hospital, Richmond and Emergency Hospital, Washington, where he continued his training during 1929 as Assistant Chief Resident Physician in Medicine and Surgery.

From 1929 to 1938 Dr. Gladding served as a medical examiner for the Pennsylvania Railroad. He was also in private practice until World War II. From 1944 to 1946 he served in the Medical Corps of the U. S. Army, attaining the rank of Lieutenant Colonel. In 1946 he was appointed to the Appeals Board of the Veterans Administration. He retired in 1965.

Dr. Gladding was elected to Active membership in the Medical Society of the District of Columbia in 1949 and was a member of the American Medical Association. He belonged to the Evergreen Masonic Lodge of Cape Charles, Va.

Surviving him are his wife, Mrs. Marie McCathran Gladding; a daughter, Jean M. Gladding; and a brother, William T. Gladding, of New Church, Va.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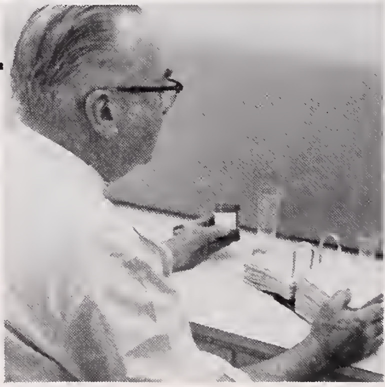

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Book Reviews

Surgery of the Aged and Debilitated Patient. Edited by John H. Powers, M.D. Price, $19.00. Pp. 611, illustrated. Philadelphia: Saunders, 1968.

After one discards the idea that age and debility are synonyms some enjoyable reading is found in this volume. The editor has leaned heavily on names and as if that were not enough he has leaned much more on the Harvard-New England axis. This is reminiscent of the proposed title for an all-time best seller, "Lincoln's Doctor's Dog."

The first half of the book is devoted to essentially basic science and is loaded with facts, data and graphs which document some of the concepts we have suspected clinically. I recommend a thorough reading of this section if one expects to do a lot of geriatric surgery.

Roughly, the second half is devoted to clinical surgery and is written well and presented in the same manner, but it is essentially the same material rehashed from the *Surgical Clinics of North America* and like organs by the same contributors.

As often happens in a book of this type controversy exists within its covers. One chapter admonishes to avoid standard incisions while another suggests using incisions that the operator is "at home with." Another contributor suggests pyloroplasty and vagotomy for ulcer while another is still with the "empiric gastrectomy" (modernization of the blind gastrectomy). The chapter on Orthopedics is superficial and is probably handled better in most standard textbooks. Another apparent error is the suggestion that primary resection and reconstitution of the bowel is acceptable for sigmoid volvulus.

The final chapter is devoted to the surgeon's perennial antagonist, the pathologist, and his unending search for causes of death. His discussion revolves around a series of 1,330 operations with 93 deaths, of which OVER half were due to our surgical errors.

Despite shortcomings, I commend this book to all who operate on the aged. The student, intern, and generalist may choose to read parts, but their time could be spent more wisely in other volumes.

Leroy Jones, M.D.

Ciba Foundation Symposium: Cell Differentiation. Edited by A. V. S. de Reuck and Julie Knight. Price, $12.00. Pp. 257, with 43 illustrations. Boston: Little, 1967.

This is a new symposium in the Ciba Foundation Series and it deals with the biologic phenomenon of cell differentiation. It is presented by 24 experimental biologists who are recognized world-authorities in the field.

The first paper introduces the subject, presenting a general review of the present concepts of the nature of cell differentiation. A basic paper on single cell properties which follows includes the discussion of the structure of cell membranes, micelle formation, and cell membrane formation and development. The biochemical changes during fertilization and early embryonic development in a number of animal species are then presented, with special reference to DNA and RNA synthesis in the oocyte, zygote and gastrula stages of the sea urchin.

Other topics discussed are the effects of nuclear transplantation on cell differentiation in the egg of the frog *Xenopus laevis*; studies on the feedback regulation of erythropoiesis in lethally irradiated mice; information transfer between cells as a factor in controlling embryonic development; and the problem of the chemical nature of embryonic inducers. There is an excellent paper on the regulation of gene expression in animal cells and another on the control of synthetic activity during cellular development following fertilization.

The last 3 papers deal directly with the interesting subject of cytodifferentiation and carcinogenesis in animal model systems.

This book is written by experimental biologists and should be of interest mainly to the basic scientists and others whose interests lie in the fields of cytochemistry and cancer research.

Jorge B. Morales, M.D.

Ciba Foundation 100th Symposium: Health of Mankind. Edited by Gordon Wolstenholme and Maeve O'Connor. Price, $12.00. Pp. 297, with 38 illustrations. Boston: Little, 1967.

In March, 1967, 28 men and women joined together in the Ciba Foundation's comprehensive Symposium on the Health of Mankind. They were doctors, city planners, biological scientists, and engineers, all expert and well-known.

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Book Reviews (*Continued*)

specific for our age, which will break us if we cannot cope with them.

The major health problems of the world are examined: the most important communicable diseases, maternal and child care, animal health and its implications for man, the mental and behavioral disorders, occupational disease and accidents, and the geographical pathology of the diseases which are the great killers of the developed countries of the Western World. These considerations are followed by an examination of the conditions of our time in history which aggravates the health problems of the world. Some of these constitute health problems in themselves and endanger the survival of the human race. The population explosion in the face of the declining death rates, malnutrition and starvation, the pollution of water and air, the dehumanization of the cities, and other problems of urbanization are included. There is a discussion of the political and social, ethnical and national limitations to the solutions of health problems. There are stimulating suggestions of routes toward resolution of these overwhelming problems, explorations of the world resources of trained health personnel, and the role of education and clinical research, as well as the possibility of the creation of a world health service as a first step towards a world society.

Of course, the symposium does not offer perfect coverage of its subject matter. The topic is too vast for this to be possible or practical. However, it is a magnificent attempt to look at what must be looked at now. This reviewer strongly recommends it as essential reading for the physician, the psychologist, the sociologist, and the educator, for the medical student and the nurse, for all who are involved in the health of mankind.

CLAIRE COBB, M.D.

Ciba Foundation Study Group No. 27. The Human Adrenal Cortex: Its Function Throughout Life. In Honour of Professor Dr. h.c.F. Verzár. Edited by G. E. W. WOLSTENHOLME AND RUTH PORTER. Price, $4.25. Pp. 146, with 26 illustrations. Boston: Little, 1967.

This volume covers the conference organized by the

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CIBA Foundation in 1966 to honor Professor h.c.F. Verzár on his eightieth birthday, and is concerned with many aspects of adrenal function in its physiological role. An impressive list of individual speakers, including Peter Forsham, Alfred Jost, L. J. Soffer, I. H. Mills, and A. Prader, covered various areas of adrenal physiology.

There is much value in this volume for the academician and the student interested in adrenal function in various life situations. The book contains the strength of each author's individual specialization but lacks general cohesiveness and relates poorly to general aspects of endocrinology. The most valuable aspects of the conference come in the discussion sections, where the remarks are cogent and stimulating. Much of the formal presentations review well known facts at great length.

The conference, unfortunately, focuses only briefly on newer developments in this field, such as the effects of cortisone on lysozymes and the interrelationships of growth hormone and cortisone.

Because of its narrow range and often too detailed summaries, this book cannot be recommended to the average physician or student.

W. Tabb Moore, M.D.

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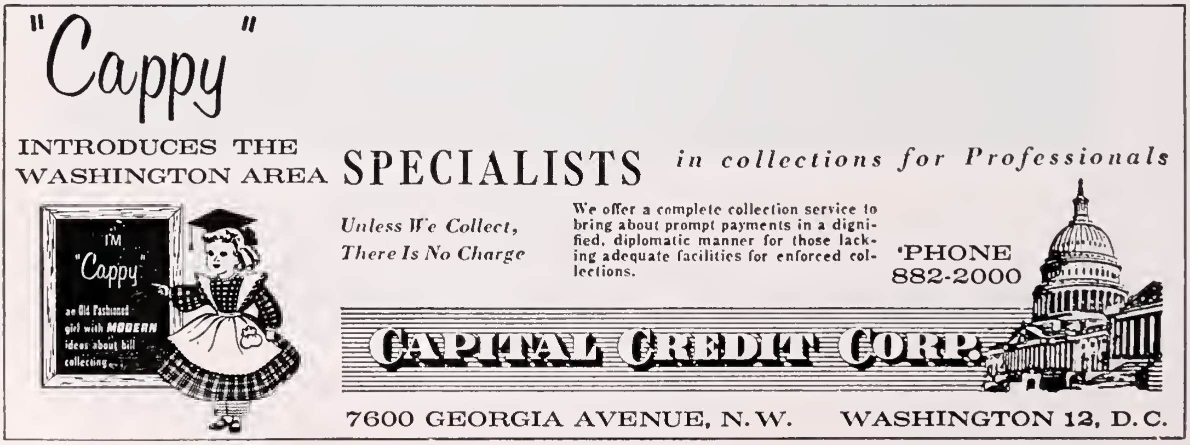
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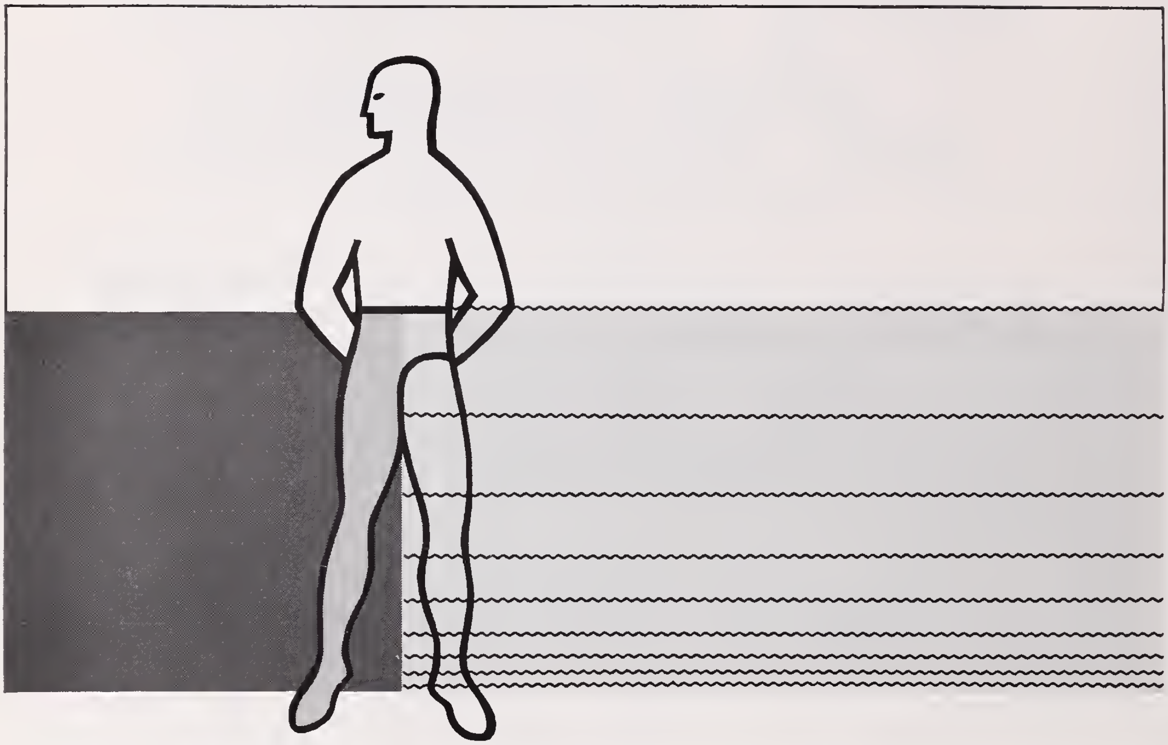

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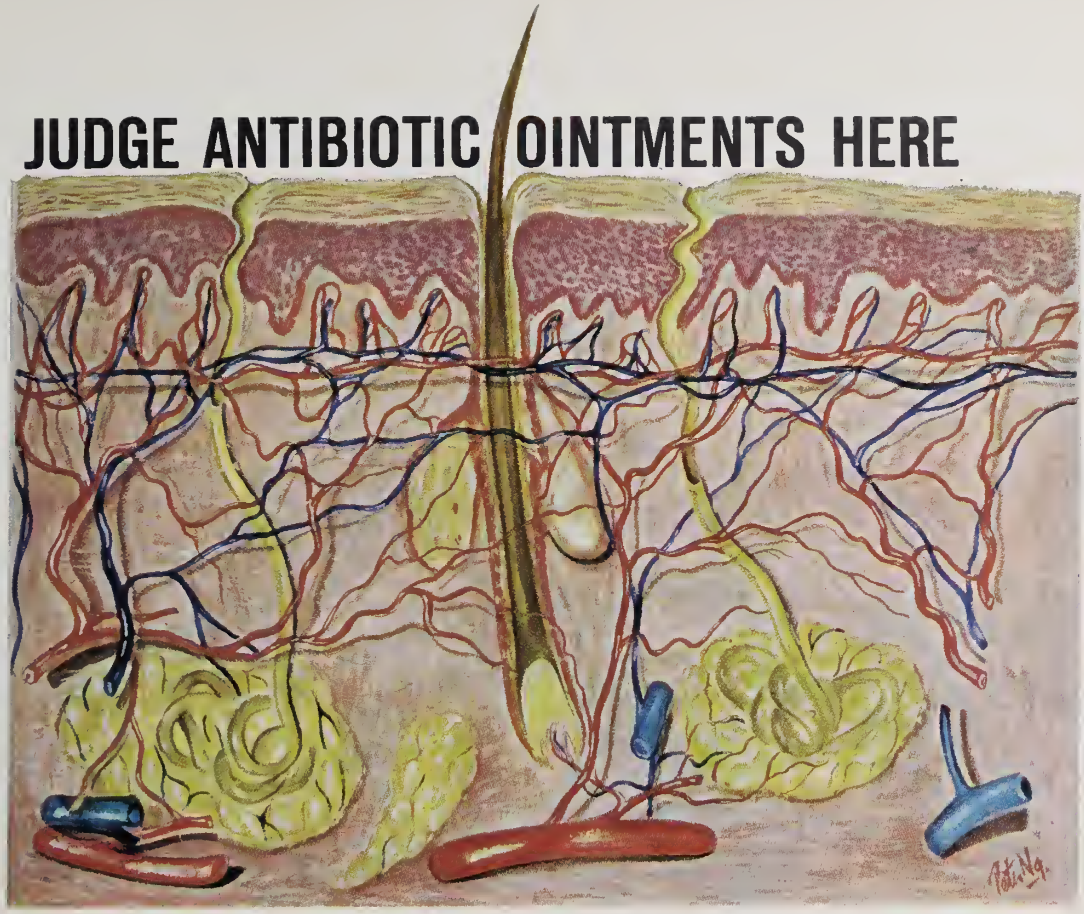

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Caution: As with other antibiotic preparations, prolonged use may result in overgrowth of nonsusceptible organisms and/or fungi. Appropriate measures should be taken if this occurs. Articles in the current medical literature indicate an increase in the prevalence of persons allergic to neomycin. The possibility of such a reaction should be borne in mind.
Contraindications: This product is contraindicated in those individuals who have shown hypersensitivity to any of its components.
Supplied: Tubes of 1 oz., ½ oz. with applicator tip, and ⅛ oz. with ophthalmic tip.
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trigeminal neuralgia

Warning
Fatal cases of aplastic anemia have been reported following treatment with Tegretol. Agranulocytosis, thrombocytopenia and transitory leukopenia have also been observed.
Complete blood and platelet counts should be done prior to and at regular intervals during treatment with the drug (see recommendations under "Important Note and Precautions") to help in the early detection of serious bone marrow injury. Abnormalities in initial blood tests should rule out use of the drug. Also, patients should be made aware of such early toxic signs of a potential hematologic problem as fever, sore throat, mouth ulcers, easy bruising and petechial or purpuric hemorrhage. Should such signs appear, the patient should be advised to discontinue the drug and to report to the physician immediately.

Indication Tegretol is indicated in the treatment of the pain associated with true trigeminal neuralgia. It is not a simple analgesic and should never be administered for relief of trivial facial aches or pains.
Contraindication Do not use in those with a known sensitivity to any tricyclic compound or in those being treated with M.A.O.I. agents. As long a period as possible should elapse before using Tegretol in patients who have been treated with M.A.O.I.'s, with a minimum of 7 days. In such cases, initial dosage should be low and the patient's reaction to gradual increments closely observed.
Important Note and Precautions Familiarity with the clinical symptoms which lead to an accurate diagnosis of true trigeminal neuralgia and with the complete prescribing information, careful patient selection, a thorough examination before treatment and close patient supervision throughout the treatment period are essential to the safe and effective use of this drug.
Where feasible, Tegretol should not be used in conjunction with any potent drug which may increase the possibility of toxic reactions.
In pregnancy, the drug should not be prescribed during the first trimester and thereafter only to patients in whom the clinical situation warrants the potential risk. It should not be administered to nursing mothers.
Patients with increased intraocular pressure should be closely observed during treatment with this drug because of its anticholinergic effect.
Because of the drug's relationship to other tricyclic compounds, the possibility of activation of latent psychosis and, in the elderly, of confusion or agitation, should be considered.
Dizziness and drowsiness may occur and patients should be cautioned about the hazards of operating machinery or automobiles and of engaging in other hazardous tasks.
Use cautiously in patients with a history of coronary artery disease, organic heart disease, congestive failure or liver disease.
Before initiating therapy, the following laboratory procedures should be performed:
1. Complete blood and platelet counts which, if abnormal, should rule out the use of the drug.
2. Baseline evaluations of liver function.
During treatment with Tegretol, the following laboratory procedures should be performed:
1. Complete blood and platelet counts should be done at intervals of one week during the first month of drug treatment, every two weeks during the second and third months, and at monthly intervals thereafter for as long as the patient is taking the drug. A trend toward a decreasing white blood cell count should suggest a dosage reduction and more frequent laboratory and clinical evaluations. Should this trend continue, the drug should be discontinued.
2. Liver function tests must be performed at regular intervals during treatment with this drug since liver damage may occur during therapy. The drug should be discontinued immediately in cases of aggravated liver dysfunction or active liver disease.
3. Periodic eye examinations, including slit-lamp, funduscopy and tonometry, are recommended for patients being treated with this drug since many phenothiazines and related drugs have been shown to cause eye changes.
4. Complete urinalysis and BUN should be done on patients treated with Tegretol because of observed renal dysfunction.
Adverse Reactions Dizziness, drowsiness, unsteadiness on the feet, nausea, vomiting, aplastic anemia, transitory leukopenia, agranulocytosis, eosinophilia, leukocytosis, thrombocytopenia, purpura, abnormalities in liver function tests, cholestatic and hepatocellular jaundice, urinary frequency, acute urinary retention, oliguria with elevated blood pressure, albuminuria, glycosuria, elevated BUN, microscopic deposits in the urine, impotence, disturbances of coordination, confusion, headache, fatigue, blurred vision, transient diplopia and oculomotor disturbances, speech disturbances, abnormal involuntary movements, peripheral neuritis and paresthesias, depression with agitation, talkativeness, nystagmus, tinnitus, paralysis and other symptoms of cerebral arterial insufficiency, pruritic and erythematous rashes, urticaria, Stevens-Johnson syndrome, photosensitivity reactions, alterations in skin pigmentation, exfoliative dermatitis, alopecia, diaphoresis, recurrence of thrombophlebitis, erythem multiforme and nodosum, aggravation of disseminated lupus erythematosus, gastric distress, abdominal pain, diarrhea, constipation, anorexia, dryness of the mouth and pharynx, glossitis, stomatitis, fever, chills, adenopathy, lymphadenopathy, aching joints and muscles, leg cramps, conjunctivitis, left ventricular failure, aggravation of hypertension, hypotension, syncope and collapse, edema, aggravation of coronary artery disease and congestive heart failure. (Whether these cardiovascular effects are drug-related is not known. However, some of these complications have resulted in fatalities.) The necessity for discontinuing the drug should be dictated by the gravity and severity of the adverse reactions.
Dosage and Administration The drug should always be taken with meals, if possible.
Initial: One-half tablet (100 mg.) b.i.d. on the first day. Thereafter, the dose should be increased in one-half tablet (100 mg.) increments every 12 hours until freedom from pain is achieved. To relieve pain, between 200 mg. and 1200 mg. per 24 hours may be necessary.
Maintenance: Initial control of pain can be maintained in most patients with a dose of 400 mg. to 800 mg. daily. Maintenance doses may range between 200 mg. and 1200 mg. daily.
At least once every 3 months during treatment period attempts should be made to discontinue the drug or to reduce the dose to the minimum effective level.
Availability Round, white, single-scored tablets of 200 mg. in bottles of 100 and 1000. (B)46-820-A
For complete details, please see Prescribing Information.

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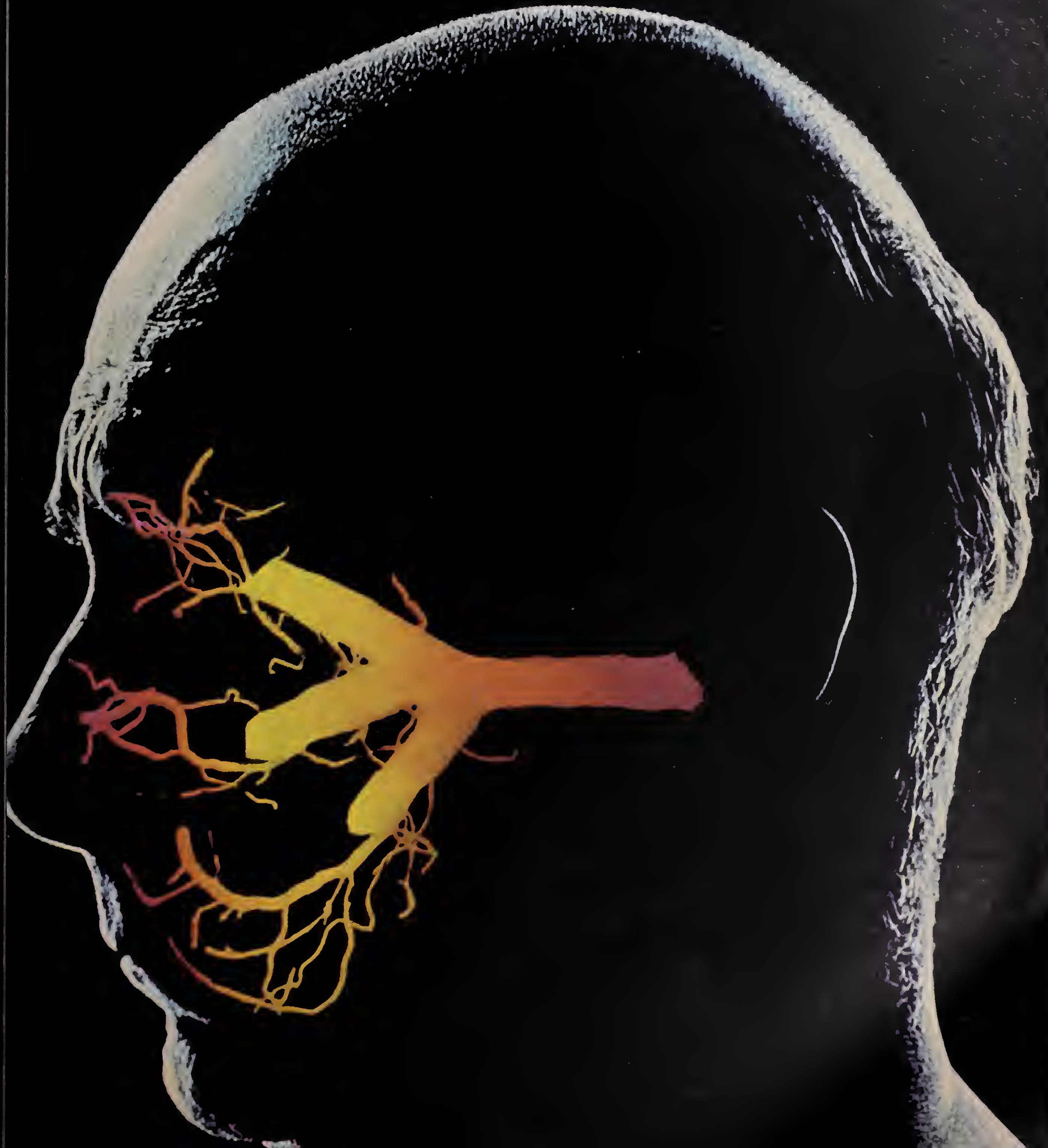

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Indications: Hypertension and many types of edema involving retention of salt and water.
Contraindications: Hypersensitivity and most cases of severe renal or hepatic disease.
Warning: With the administration of enteric-coated potassium supplements, which should be used only when adequate dietary supplementation is not practical, the possibility of small bowel lesions (obstruction, hemorrhage, and perforation) should be kept in mind. Surgery for these lesions has frequently been required and deaths have occurred. Discontinue enteric-coated potassium supplements immediately if abdominal pain, distention, nausea, vomiting, or gastrointestinal bleeding occur.
Use with caution in pregnant patients. Since the drug may cross the placental barrier, adverse reactions which may occur in the adult (thrombocytopenia, hyperbilirubinemia, altered carbohydrate metabolism, etc.) are potential problems in the newborn.
Precautions: Antihypertensive therapy with this drug should always be initiated cautiously in postsympathectomy patients and in patients receiving ganglionic blocking agents or other potent antihypertensive drugs, or curare. Reduce dosage of concomitant antihypertensive agents by at least one-half. Barbiturates, narcotics or alcohol may potentiate hypotension. Because of the possibility of progression of renal damage, periodic determination of the BUN is indicated. Discontinue if the BUN rises or liver dysfunction is aggravated. Hepatic coma may be precipitated.
Electrolyte imbalance, sodium and/or potassium depletion may occur. If potassium depletion should occur during therapy, the drug should be discontinued and potassium supplements given, provided the patient does not have marked oliguria.
Take special care in cirrhosis or severe ischemic heart disease and in patients receiving corticosteroids, ACTH, or digitalis. Salt restriction is not recommended.
Adverse Reactions: Nausea, gastric irritation, vomiting, anorexia, constipation and cramping, dizziness, weakness, restlessness, hyperglycemia, hyperuricemia, headache, muscle cramps, orthostatic hypotension, aplastic anemia, leukopenia, thrombocytopenia, agranulocytosis, impotence, dysuria, transient myopia, skin rashes, urticaria, purpura, necrotizing angiitis, acute gout, and pancrec titis when epigastric pain or unexplained G.I. symptoms develop after prolonged administration. Other reactions reported with this class of compounds include: jaundice, xanthopsia, paresthesia, and photosensitization.
Average Dosage: 50 or 100 mg. with breakfast daily or 100 mg. every other day.
Availability: White, single-scored tablets of 100 mg. and aqua tablets of 50 mg., in bottles of 100 and 1000.
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Indications: Indicated when anxiety, tension and apprehension are significant components of the clinical profile.

Contraindications: Patients with known hypersensitivity to the drug.

Warnings: Caution patients about possible combined effects with alcohol and other CNS depressants. As with all CNS-acting drugs, caution patients against hazardous occupations requiring complete mental alertness (*e.g.*, operating machinery, driving). Though physical and psychological dependence have rarely been reported on recommended doses, use caution in administering to addiction-prone individuals or those who might increase dosage; withdrawal symptoms (including convulsions), following discontinuation of the drug and similar to those seen with barbiturates, have been reported. Use of any drug in pregnancy, lactation, or in women of childbearing age requires that its potential benefits be weighed against its possible hazards.

Precautions: In the elderly and debilitated, and in children over six, limit to smallest effective dosage (initially 10 mg or less per day) to preclude ataxia or oversedation, increasing gradually as needed and tolerated. Not recommended in children under six. Though generally not recommended, if combination therapy with other psychotropics seems indicated, carefully consider individual pharmacologic effects, particularly in use of potentiating drugs such as MAO inhibitors and phenothiazines. Observe usual precautions in presence of impaired renal or hepatic function. Paradoxical reactions (*e.g.*, excitement, stimulation and acute rage) have been reported in psychiatric patients and hyperactive aggressive children. Employ usual precautions in treatment of anxiety states with evidence of impending depression; suicidal tendencies may be present and protective measures necessary. Variable effects on blood coagulation have been reported very rarely in patients receiving the drug and oral anticoagulants; causal relationship has not been established clinically.

Adverse Reactions: Drowsiness, ataxia and confusion may occur, especially in the elderly and debilitated. These are reversible in most instances by proper dosage adjustment, but are also occasionally observed at the lower dosage ranges. In a few instances syncope has been reported. Also encountered are isolated instances of skin eruptions, edema, minor menstrual irregularities, nausea and constipation, extrapyramidal symptoms, increased and decreased libido—all infrequent and generally controlled with dosage reduction; changes in EEG patterns (low-voltage fast activity) may appear during and after treatment; blood dyscrasias (including agranulocytosis), jaundice and hepatic dysfunction have been reported occasionally, making periodic blood counts and liver function tests advisable during protracted therapy.

Usual Daily Dosage: Individualize for maximum beneficial effects. *Oral*—Adults: Mild and moderate anxiety and tension, 5 or 10 mg t.i.d. or q.i.d.; severe states, 20 or 25 mg t.i.d. or q.i.d. Geriatric patients: 5 mg b.i.d. to q.i.d. (See Precautions.)

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August
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Hazards of High Parity
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Angiographic Study of Pancreas
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Acute Pulmonary Edema
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SIDE EFFECTS: Due mainly to anticholinergic action and usually at high dosage. They may include dryness of the mouth, tachycardia, palpitation, urinary hesitancy or retention, blurred vision, dilatation of the pupil, increased ocular tension, weakness, nausea, vomiting, headache, dizziness, constipation, and drowsiness. Infrequently, mental confusion in the elderly, urticaria or other dermatoses. Side effects are usually eliminated by reduction in dosage. Two cases of aplastic anemia, with no established causal relationship, have been reported.

DOSAGE: **INJECTABLE** — Average adult dose: one ampul, 2 cc. (60 mg. orphenadrine citrate) I.M. or I.V. May be repeated every 12 hours. Relief may be maintained with one NORFLEX tablet b.i.d. **TABLETS** — Two tablets per day for adults, one in the morning, one in the evening. Each tablet contains 100 mg. orphenadrine citrate.

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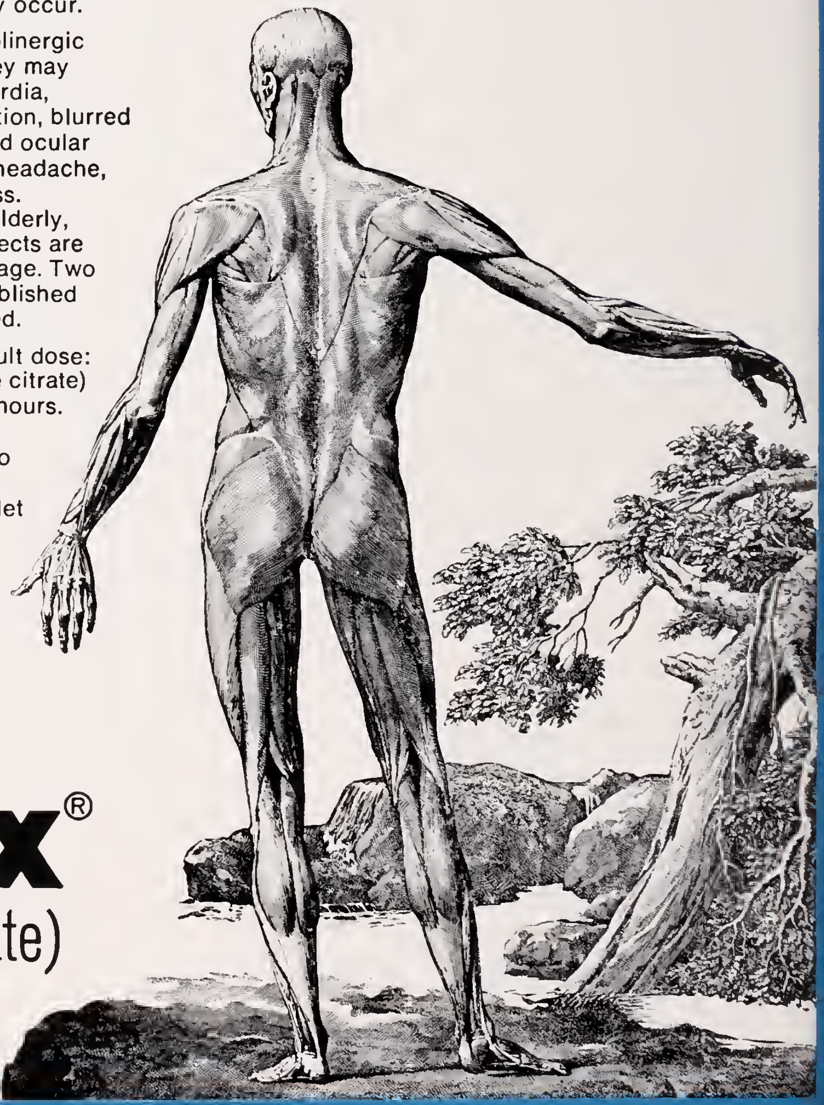

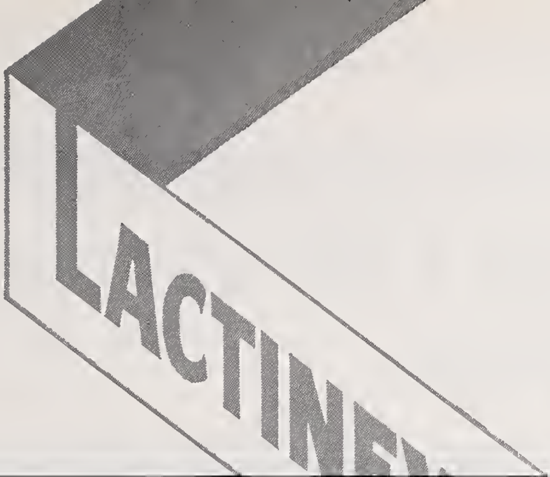

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References:

(1) Siver, R. H.: CMD, *21*:109, September 1954. (2) Frykman, H. H.: Minn. Med., *38*:19-27, January 1955. (3) McGivney, J.: Tex. State Jour. Med., *51*:16-18, January 1955. (4) Quehl, T. M.: Jour. of Florida Acad. Gen. Prac., *15*:15-16, October 1965. (5) Weekes, D. J.: N.Y. State Jour. Med., *58*:2672-2673, August 1958. (6) Weekes, D. J.: EENT Digest, *25*:47-59, December 1963. (7) Abbott, P. L.: Jour. Oral Surg., Anes., & Hosp. Dental Serv., 310-312, July 1961. (8) Rapoport, L. and Levine, W. I.: Oral Surg., Oral Med. & Oral Path., *20*:591-593, November 1965.

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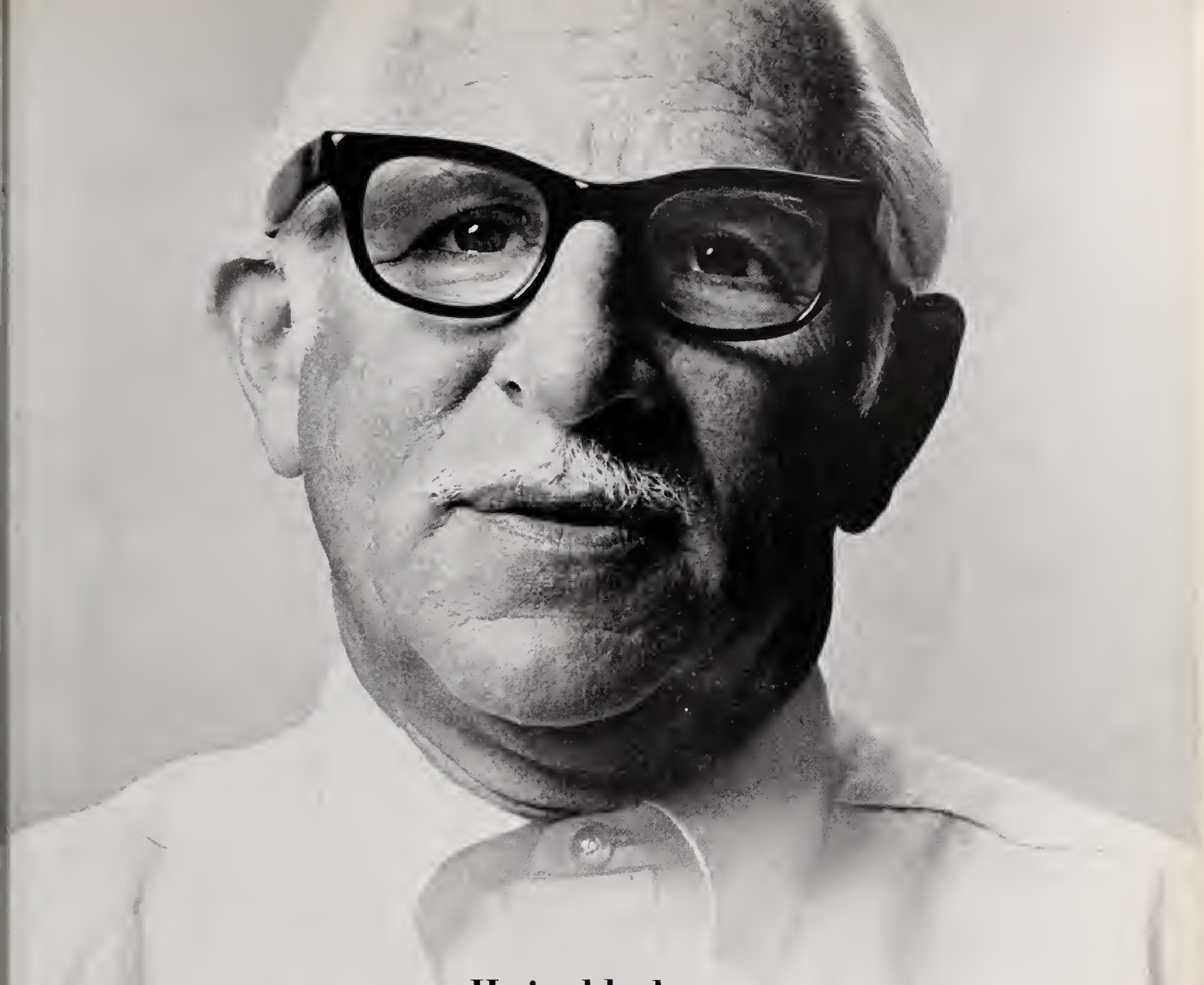

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ning: In renal impairment, usual doses may lead to excessive accumu- on and liver toxicity. Under such conditions, lower than usual doses indicated, and, if therapy is prolonged, serum level determinations be advisable. A photodynamic reaction to natural or artificial sun- t has been observed. Small amounts of drug and short exposure may luce an exaggerated sunburn reaction which may range from ery- na to severe skin manifestations. In a smaller proportion, photo- gic reactions have been reported. Patients should avoid direct osure to sunlight and discontinue drug at the first evidence of skin omfort. Necessary subsequent courses of treatment with tetracy- es should be carefully observed.

autions: Overgrowth of nonsusceptible organisms may occur. Constant observation is essential. If new infections appear, appropriate measures should be taken.

In infants, increased intracranial pressure with bulging fontanels has been observed. All signs and symptoms have disappeared rapidly upon cessation of treatment.

Side Effects: Gastrointestinal system—anorexia, nausea, vomiting, diarrhea, stomatitis, glossitis, enterocolitis, pruritus ani. Skin—maculopapular and erythematous rashes; a rare case of exfoliative dermatitis has been reported. Photosensitivity; onycholysis and discoloration of the nails (rare). Kidney—rise in BUN, apparently dose related. Transien increase in urinary output, sometimes accompanied by thirst (rare). Hypersensitivity reactions—urticaria, angioneurotic edema, anaphylaxis. Teeth—dental staining (yellow-brown) in children of mothers given this drug during the latter half of pregnancy, and in children given the drug during the neonatal period, infancy and early childhood. Enamel hypoplasia has been seen in a few children. If adverse reaction or idiosyncrasy occurs, discontinue medication and institute appropriate therapy. Average Adult Daily Dosage: 150 mg q.i.d. or 300 mg b.i.d. Should be given 1 hour before or 2 hours after meals, since absorption is impaire by the concomitant administration of high calcium content drugs, food and some dairy products. Treatment of streptococcal infections shoul continue for 10 days, even though symptoms have subsided.

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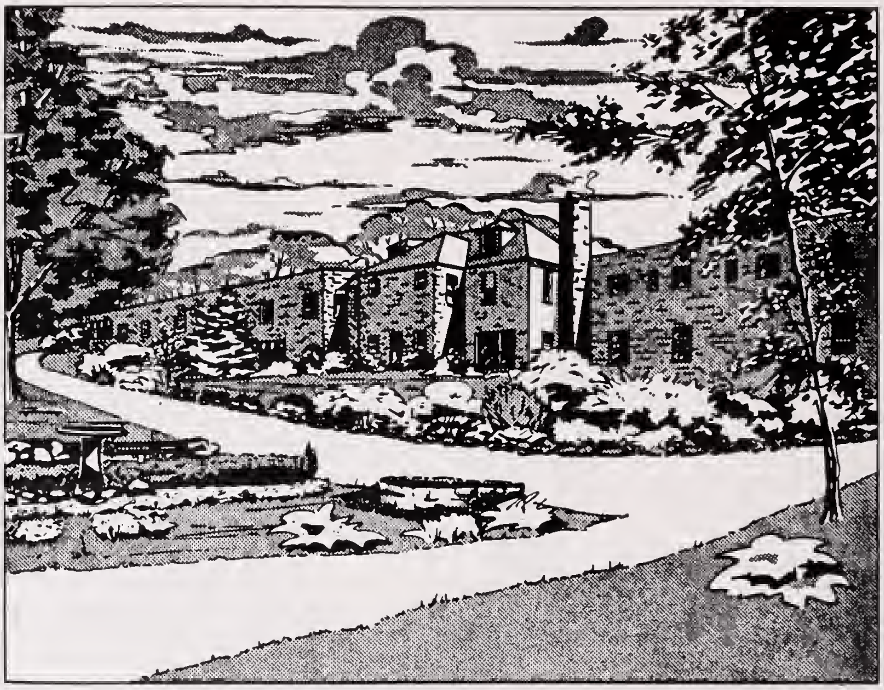

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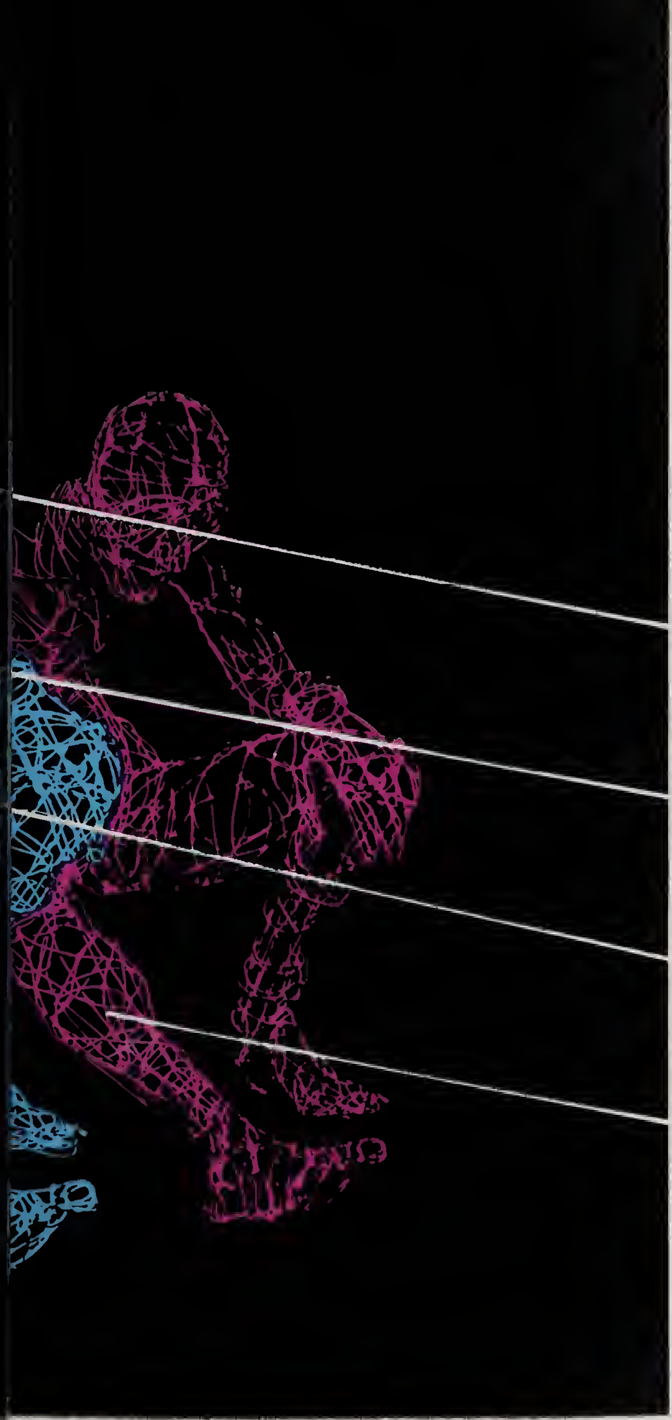

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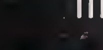
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Book Reviews

Pathology of Infancy and Childhood. John M. Kissane, M.D., and Margaret G. Smith, M.D. Price, $39.50. Pp. 1082, with 703 illustrations. St. Louis: Mosby, 1967.

This is a very readable, amply illustrated, well-written and well-organized text on pediatric pathology. The material is divided into 14 sections and 42 chapters. At the end of each chapter is an excellent list of pertinent references, many recent. The organization is that of a conventional text of systemic pathology except for the first 2 sections which deal with diseases related to growth and intermediary metabolism and with infectious diseases. The emphasis is on those diseases or aspects of disease which are either peculiar to or of particular importance in infancy and childhood. There are very good chapters on diseases of the lungs and disorders of sexual differentiation as well as thorough discussions of many important entities such as fibrocystic disease of the pancreas. The inclusion of a brief review of normal organ development in most sections and chapters is most felicitous.

Although broad in scope, this text cannot be considered a complete treatise on pediatric pathology. The omission of a section on placental pathology is particularly regretted. One also wishes that more attention had been paid to neonatal pathology and that the discussions of lesions of the newborn infant had been brought together in a single chapter or section. Although not the result of oversight on the part of the authors, this is a significant limitation in terms of the user of the book.

The illustrations are generally of high quality. Not all, however, are clearcut, and some of the accompanying legends are either insufficient or ambiguous. Occasional errors, mostly typographical, are found. The suggested retail price of $39.50 is rather high. One is concerned that the continued rise in the price of medical books will deter many physicians from expanding their professional libraries.

Notwithstanding the above shortcomings, this is, all in all, a fine book. It is recommended to pathologists-in-training, general pathologists, and to pediatricians with some background knowledge of general pathology. Every hospital with a training program in pathology should have it on its shelves.

Stephen G. Kent, M.D.

Atlas of Roentgenographic Positions, 3rd ed. Vinita Merrill. Price per 3-volume set, $46.50. Pp. 878, illustrated. St. Louis: Mosby, 1967.

The third edition of the "Atlas of Roentgenographic Positions" has been completely rewritten and expanded from 2 to 3 volumes. The extra volume is necessitated by the expansion of old material, the addition of many new, non-routine techniques, and a new chapter on protection against ionizing radiation. A separate volume is now devoted to the skull and related anatomical parts.

The smaller print size, rearrangement of many of the pictures, and the increase in clarity of the radiographic reproductions add to the readability and esthetic appeal of the third edition.

The new sections to the third edition are too numerous to list in detail, but the section on the eye including the anatomy and foreign body localization, the section on the jugular foramen and hypoglossal canal, the section on arthrography of the knee, and the section on the male reproduction tract deserve special mention for the clarity and completeness of presentation.

Many new radiographic positions are included in the revised sections including those for the glenohumeral joint, the subtalar joint, hiatal hernia, and others. New techniques have been added including drip infusion pyelography, the chain test used in evaluation of the female bladder for stress incontinence, intubation technique for the small bowel, and others. The section on mammography has been expanded in keeping with the surge in popularity of this examination.

The thoroughness of the third edition is almost incredible considering the voluminous material covered. But with the 10-year hiatus between editions, the little emphasis given to the head unit, failure to include the rotation chair in pneumoencephalography, and the very abbreviated section on arteriography leave a rather significant void. More captions beneath the radiographs and more use of line drawings would add clarity, especially for the student of radiology.

The "Atlas of Roentgenographic Positions" has been the classic reference text in this field since the first printing and with the third edition will retain this deserved reputation.

Larry Stanton, M.D.

Ciba Foundation Volume. Communication in Science: Documentation and Automation. Edited by Anthony deReuck and Julie Knight. Price, $12.50. Pp. 274, with 17 illustrations. Boston: Little, 1967.

This book deals with the growth of the scientific literature—the so-called "information explosion." It is

(*Continued on page 17*)

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Book Reviews

(Continued from page 15)

the record of a symposium in which scientists and communications experts exchanged ideas on the subject.

One speaker made this keynote statement: "This problem—that of keeping abreast of advancing knowledge in one's subject—is only one of the 3 basic problems we have to face, the others being the retrieval of information already existing and the conveyance of relevant information from one science to the practitioners of another."

To the practitioners of medicine this has too familiar a sound to require elaboration. The literature is mountainous, and keeping up with it is impossible for most of us.

What, then, is the solution? All of the contributors except one seem to be of one mind: Ways must be found to effect better storage, retrieval and dissemination of essentially all published scientific information. Several attempts to do this are described, including the National Library of Medicine's MEDLARS (Medical Literature Analysis and Retrieval System) Program. They all involve the use of computers and a "network" of institutions which utilize the systems.

None of the proposals except one seems likely to help the medical practitioner very much, at least in the near future. The exception is a suggestion made by Dr. D. J. De Solla Price of the Department of the History of Medicine and Science of Yale University. He first made the observation that, even in the day of Faraday and Franklin, half of all the scientific material ever published had appeared in the last 15 years. He then suggested that most of it, today as in the past, wasn't worth the effort to store and retrieve. He proposed that much of the literature be treated "as perishable goods to be discarded like the daily newspaper and only preserved in a few great libraries for the benefit of sentimental historians." He proposes that "a paper the size and shape of the *Financial Times* or *Wall Street Journal* might carry the daily world total non-archival production over all sciences, technology and medicine and have room over for a bonus page of social and political news of science too."

Which of the 2 approaches described has more merit remains to be seen. It is of note, however, that the most successful medical publications to be started in recent years are of the newsmagazine and newspaper type. What doctors seem to want is not so much storage and retrieval of published materials but more screening and interpretation of the research discoveries they can use in their practices.

The book itself is not likely to be of interest to most physicians. Those interested in the production and dissemination of medical information, however, will find it a thoughtful attempt to solve a growing problem.

LOUIS F. RITTELMEYER, JR., M.D.

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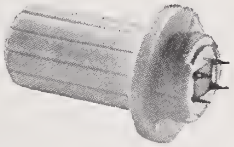

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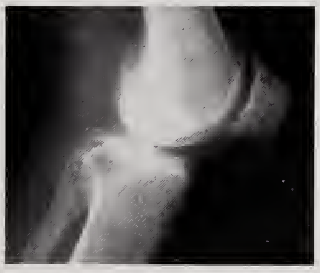

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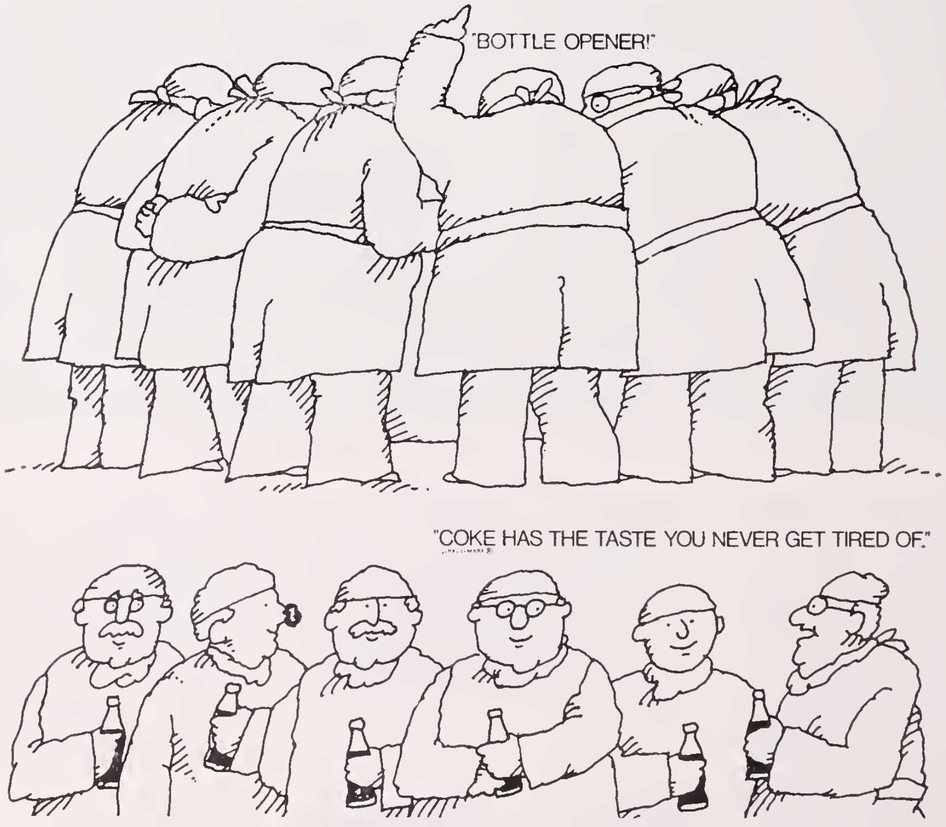
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►NATIONAL & REGIONAL MEETINGS

August 25–31: International Congress of Physiological Sciences. Washington. Address: Secretariat, International Congress of Physiological Sciences, 9650 Rockville Pike, Bethesda, Md. 20014.

September 1–6: American Society of Hematology. Waldorf-Astoria, New York. Address: Dr. J. F. Mustard, Department of Pathology, McMaster University, Hamilton, Ontario.

September 5–7: American Association of Obstetricians and Gynecologists. The Homestead, Hot Springs, Va. Address: Dr. Robert B. Wilson, Executive Secretary, 200 First Street, S.W., Rochester, Minn. 55901.

September 6–7: Cardiovascular Symposium sponsored by Tidewater Heart Association and Council on Clinical Cardiology, American Heart Association. Lake Wright Motel, Virginia Beach, Va. Address: Dr. James D. Price, Chairman, 523 Boush Street, Norfolk, Va. 23510.

September 13–20: American Academy of General Practice. Las Vegas, Nevada. Address: Mr. Mac F. Cahal, Executive Director, Volker Blvd. at Brookside, Kansas City, Mo. 64112.

September 15–20: International Congress on Alcohol and Alcoholism. Shoreham Hotel, Washington. Address: Secretariat Office, 1130 17th Street, N.W., Washington, D. C. 20036.

September 19–22: American Medical Writers' Association. Mayflower Hotel, Washington. Address: Mr. Stephen T. Donohue, Treasurer, 420 Lexington Avenue, New York, N. Y. 10017.

September 25–27: American Association of Medical Clinics. The Roosevelt, New Orleans. Address: Dr. Edward M. Wurzel, Executive Director, 421 King Street, Alexandria, Va. 22314.

September 30–October 1: AMA Congress on Occupational Health. Waldorf-Astoria Hotel, New York. Address: AMA Department of Occupational Health, 535 North Dearborn Street, Chicago, Ill. 60610.

October 4–8: International Congress on Diseases of the Chest. Washington Hilton Hotel, Washington. Address: American College of Chest Physicians, 112 East Chestnut Street, Chicago, Ill. 60611.

October 14–18: American College of Surgeons. Dennis Hotel, Atlantic City, N. J. Address: Dr. John Paul North, 55 East Erie Street, Chicago, Ill. 60611.

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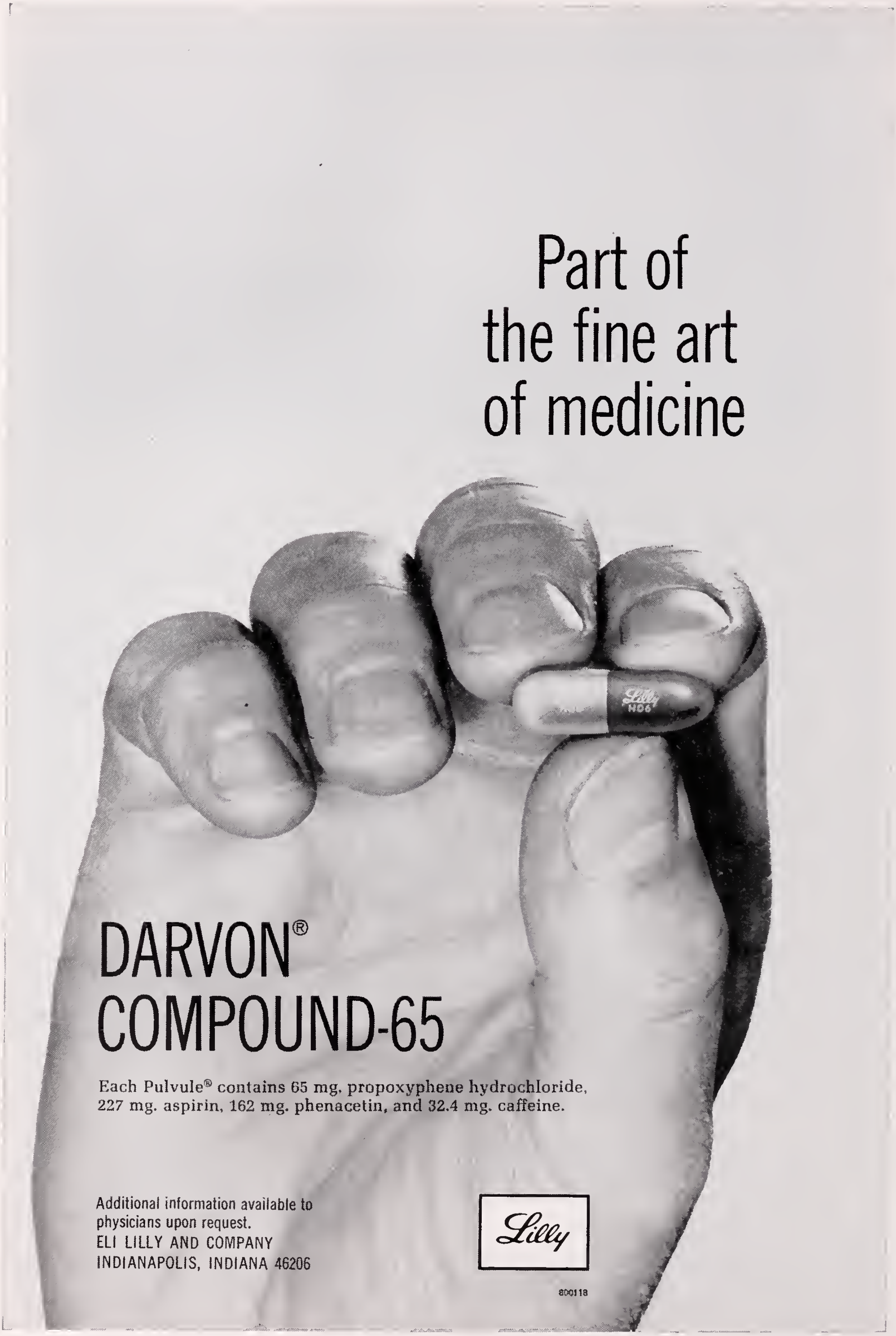

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Ascariasis*

DENNIS M. GREENBAUM, M.D.†‡

IT HAS BEEN ESTIMATED that about 25 per cent of the world population and about 1 to 2 per cent of the population of the United States are hosts to *Ascaris lumbricoides.*[1] In geographic distribution it is ubiquitous, Iceland being the only country which had not reported a case up to 1940.[2] In this paper, our current experience with this disease will be reviewed and its diagnosis and treatment discussed.

Classification

The classification of pathogenic worms is very confusing when the medical literature is reviewed, partly because of the unsophisticated and incomplete characterization of the animal kingdom as recently as 15 or 20 years ago. The classification of worms in general is in fact rather clear-cut.

There are 10 phyla in the animal kingdom,§ of which 3, Platyhelminthes, Nemathelminthes, and Annelida, comprise the worms. As late as 1940 all worms were classified in one phylum, Vermes.[2] Platyhelminthes and Nemathelminthes are nonsegmented worms, the former being flatworms and the latter round, and the Annelida are segmented.[3] The term, helminth, refers to any worm, although it is often used specifically to mean an intestinal worm.[4] The vast majority of parasitic worms are members of Nematoda, one of the 3 orders of Nemathelminthes (table 1). The term, nematode, is often mistakenly used to mean intestinal parasite, for within it are the hookworm, whipworm, pinworm, and ascarids. However, the filariae as well as other extra-intestinal and nonparasitic worms are also nematodes.

The ascarids make up one family in the order Nematoda. There are 2 genera of medical significance in this family: Enterobius (Oxyuris), of which *E. vermicularis* (pinworm) is the only parasite, and Ascaris. The prototype of the genus Ascaris is *A. lumbricoides,* one of the most common infectious agents in man.[1, 5, 6]

TABLE 1: Classification of Ascaris among the Nemathelminthes*

Phylum Nemathelminthes
  A. Order Nematoda
    1. Nematodes affecting the digestive tract:
      Family Ascaridae
        Ascaris species
        Enterobius (Oxyuris) species
      Family Strongylidae
        Ancylostoma species
        Necator species
      Family Trichotrachelidae
        Trichuris species
        Trichinella species
      Family Angiostomidae
        Strongyloides species
    2. Nematodes affecting the blood and lymph:
      Filariae
    3. Nematodes affecting the subcutaneous tissue:
      Dracontids
    4. Nematodes affecting other viscera
  B. Order Gordiacea
  C. Order Acanthocephala

* Adapted from Whitmore.[2]

Description

Ascaris lumbricoides was first described by Linnaeus in 1758.[2] It has a thick body and oral papillae which characterize it as a member of the family Ascaridae. The worm may be reddish-brown or grayish-yellow and is pointed at both ends. The head is small and has 3 finely-toothed lips, the dorsal having 2 papillae, and the 2 ventrolateral having 1 each at the base.

Ascaris may be male or female. The female is usually 8 to 10 inches by 1/5 inch, and the vulva may be found in the anterior third of the worm.

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*Based on clinical experience under the direction of Julie Wershing, M.D., Agnes Hardecker Childeren's Clinic, Nassau, Bahamas—Director: Frederic G. Burke, M.D., Georgetown University Medical Center, Washington, D. C.

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‡EDITOR'S NOTE: Author was a senior medical student at Georgetown University when he wrote this paper.

§ Protozoa, Porifera, Coelenterata, Platyhelminthes, Nemathelminthes, Annelida, Echinodermata, Mollusca, Arthropoda, and Chordata.

The male is somewhat smaller, 6 to 8 inches by ⅛ inch, with the tail rolled under it and 2 unequal spicules plainly visible. Worms much larger have often been described.

As a member of Nematoda, Ascaris is in the first order of the animal kingdom to have a complete digestive tube, separated from the body wall by a cavity, or pseudocoel. The worms have a neural ring which surrounds a primitive pharynx, from which lead dorsal and ventral nerve cords and a fine nerve-net system. An excretory canal traverses the length of the worm on either side. There is no circulatory or gas-exchange system, but movement of the worm probably generates enough current in the pseudocoel for gas and fluid exchange with the digestive tube and muscular wall.[3] Oxygen is carried by hemoglobin dissolved in the perienteric fluid of the pseudocoel.

Life Cycle

The roundworms are the most common inhabitants of the soil, and it is curious that among the ascarids are classified the pinworm, very common in and fairly well localized to the temperate zones, and Ascaris, which decreases in frequency with distance from the equator. The female Ascaris lays her eggs while in the host small intestine, and the eggs are passed either into the soil or on floors in areas where toilet and sewage control are poor or nonexistent. Ascaris eggs favor hard clay soils as compared to moist humus, and flatlands to hill country so that they are less likely to be washed away by the rains.[7] Although the egg can not tolerate severe heat, warmth is required for the fertilized egg to embryonate, and in 9 days a motile first-stage larva is produced. One moult occurs during the next 7 days, and the second-stage larva that results is infective. The egg can hatch only after being swallowed, but it will remain viable if not swallowed for months or years if tropical or semitropical environment is maintained. In these areas, especially where children have access to soil or contaminated floors on which to play, 60 to 100 per cent of children will harbor Ascaris.[6]

The second-stage larvae hatch in the duodenum or jejunum[8] and enter the intestinal mucosa, from which they have easy access to the mesenteric lymphatics and venules. They are often seen migrating through the liver, but they ultimately reach the lungs via the portal circulation and right side of the heart. The larvae bore through the alveolar capillaries into the air spaces where a second moult takes place, and the third-stage larvae migrate up the tracheobronchial tree to the epiglottis. Now the larva can be swallowed, and it becomes established in the small intestine. It takes an additional 60 to 75 days for the larvae to develop into adult worms and for these to mate and lay eggs. Each female will lay about 200,000 eggs daily, but not all of these will be fertilized. This number is so characteristic that an estimate of the number of worms harbored by the host can be made from a quantitative analysis of ova in the stool. Eggs from females who have just begun to oviposit often do not develop despite fertilization by the male.[6]

The worms may be expelled spontaneously at times per rectum, especially when there is gastrointestinal disturbance and diarrhea. Occasionally the worm will migrate upward toward the stomach and may rarely be expelled by mouth. Infrequently, the worms may collect in the appendix, gall bladder, common bile duct and pancreatic duct, or Meckel's diverticulum.[9, 10]

Predisposing Environmental Conditions

The highest prevalence of Ascaris occurs between 40° north and south latitudes. Raw fruits and vegetables often carry the embryonating ova,[6] especially where night soil is used for fertilizer. Flatland is preferable to hill country because the eggs are less likely to be washed away by rains.[7] Areas of even climate are likely to preserve viable ova as opposed to areas with yearly frosts and marked seasonal climate variations. Freezing does not allow the ova to embryonate into a first-stage larva. Sewage control alleviates many problems by carrying away infested feces, and better hygiene among primitive and poor people is likely to eliminate reinfection from the stool. In the United States highest prevalence is in the Appalachian, Ozark, and Gulf Coast states.[6]

Incidence

Although prevalence rates vary among researchers, it is generally agreed that the incidence of Ascaris infestation is very high. In many it is second to hookworm in helminth incidence, and in others it rates higher. In a survey of our records of 234 clinic visits by 33 students* at the Hardecker Children's Clinic† Ascaris ova were found in the stool on 84 occasions (35 per cent) (table 2). Of these, it was found in association with other parasites in 38 (17 per cent), and alone in 46 (19 per cent). Only 111 (47 per cent) of the examinations were negative for parasites, and 39 examinations (17 per cent) were positive for parasites other than Ascaris.

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*Students of the Montessori class at Our Lady's School, Deveaux Street, Nassau, New Providence Island, Bahamas.

†Agnes Hardecker Children's Clinic, Deveaux Street, Nassau, New Providence Island, Bahamas, affiliate of Pediatrics Department, Georgetown University Medical Center, Washington, D. C.

TABLE 2: Prevalence of Ascaris in 234 Stool Examinations*

Stool Examination	No. Children	Per cent
Normal stool	111	47
Ascaris alone	46	19
Ascaris with other parasites	38	17
Trichuris trichiura	19	8
Entamoeba histolytica	2	1
Trichuris, *E. histolytica*	8	5
Trichuris, *E. histolytica*, Giardia	2	1
Trichuris, *E. histolytica*, Giardia, Trichomonas, *Chylomastix mesneli*	2	1
Trichuris, *E. histolytica*, Trichomonas, *Chylomastix mesneli*	1	—
Trichuris, *Giardia lamblia*	2	1
Trichuris, Giardia, Trichomonas, *Chylomastix mesneli*	1	—
Trichuris, *E. histolytica*, pathogenic *Escherichia coli*	1	—
Negative for Ascaris, but others present	39	17
Trichuris trichiura	20	9
Giardia lamblia	2	1
Trichuris, Giardia	3	1
Trichuris, *E. histolytica*	1	—
E. histolytica, Giardia	2	1
Trichuris, *E. histolytica*, Giardia	3	1
Trichuris, *E. histolytica*, Giardia, *Chylomastix mesneli*	1	—
Trichuris, *E. histolytica*, Giardia, *C. mesneli*, Trichomonas	2	1
Trichuris, *E. histolytica*, *C. mesneli*	1	—
Trichuris, *E. histolytica*, *C. mesneli*, Trichomonas	1	—
Trichuris, Trichomonas	1	—
Trichuris, Giardia, *C. mesneli*	1	—
E. histolytica, *Entamoeba coli*	1	—
TOTAL	234	100

* Examinations done on 33 students in the Montessori class at Our Lady's School, Nassau, by the Agnes Hardecker Children's Clinic, Nassau, affiliate of Georgetown University Medical Center, Washington, D. C.

Of the 33 children studied (aged 4 to 7 years), 7 (21 per cent) never had a positive stool examination for ova or parasites. All the others (79 per cent) had a stool positive for Ascaris on at least one occasion. Only 3 (9 per cent) had stools positive for Ascaris and never another parasite. Twenty-three (70 per cent) had a history of harboring other parasites also. There was no child who had ever had a positive stool for any parasite that did not at some time also harbor Ascaris. In one child in whom treatment for Ascaris, *Trichuris trichiura*, and *Entamoeba histolytica* did not relieve symptoms, culture of the stool revealed pathogenic *Escherichia coli*. She was treated for this too, and in follow-up stools the following month she was found to have microscopic hematochezia and to be reinfested with Ascaris, Trichuris, and *E. histolytica*.

In a similar study at Camp Zachary Taylor, Kentucky, among 35,000 troops in 1919, 3.76 per cent were found to be harboring Ascaris and 3.27 per cent hookworm (Ancylostoma species and *Necator americanus*).[5] In a New Guinea survey of parasites in 83 patients between the ages of less than 1 year to 14 years 53 per cent were harboring Ascaris and 64 per cent hookworm.[7]

It is believed that prehistoric man was overwhelmed by Ascaris, and Stoll estimates that 644,400,000 still harbor the parasite.[1] Three million of these are within the United states.

Symptoms

The second-stage larva is not likely to cause any significant symptoms while in the intestine. Surgeons have described full-grown worms in the mesentery. These apparently developed from second-stage larvae after migrating through the bowel wall before being picked up by the venules and lymphatics which ordinarily carry the larvae through the liver to the lungs where further maturation usually takes place. One case of mesenteric thrombosis has been reported in a 9-year-old boy who was found at surgery to have 1 worm within a major mesenteric vein and another encircling and obstructing an ileal mesenteric artery from without.[10] There is at least 1 report of granulomatous peritonitis due to Ascaris.[11]

The second-stage larva passes through the liver, where it has been reported to cause liver abscesses and calcified hepatic granulomata.[12] In some cases in which the helminth has been trapped within the small portal venules in the liver it has migrated through the parenchyma to the biliary canals and descended into the gall bladder. From here it may enter the common bile duct and the pancreatic duct to cause obstruction and pancreatitis. Often the worm develops within the gall bladder and becomes too large to enter the cystic duct. It may survive here, causing symptoms of cholecystitis, but it usually dies. The presence of dead Ascaris in the gall bladder has been implicated in the formation of gall stones on at least 1 occasion.[13] In a 10-year-old Korean girl with congenital hemolytic anemia acute symptoms in the abdomen led to laparotomy for what was believed to be an acute hemolytic crisis. A splenectomy was performed, but in addition she was found to have obstruction of the common bile duct. One living and several dead adult Ascaris were removed from the biliary tree and a T-tube inserted. She was treated for ascariasis, and 14 days later the T-tube became obstructed. When it was removed it was found to have a dead adult Ascaris obstructing its lumen.[9]

The second-stage larva usually passes through the liver without consequence to the lungs, but it may cause an atypical pneumonia characterized by hemorrhage and inflammation.[6] There may be an eosinophilic infiltrate of the interstitium called Loeffler's syndrome,[8] but this is more characteristic of *Toxocara canis* and *Toxocara cati* infestations (visceral larva migrans) than of Ascaris.[6] There is 1 report of the worm developing in the tracheobronchial tree and causing death by suffocation.[14]

Usually the third-stage larva passes up the bronchial tree to the pharynx where it is swallowed. The worm may be passed from the mouth or nose at this time. The most common complaint is now mild abdominal pain, although nausea, vomiting, and anorexia are not rare. Slight fever and even profound fever in acute cases may occur, and in rare cases acute intestinal obstruction may result when a bolus of worms develops. Worms may be passed spontaneously from the rectum. Symptoms of localized enteritis with tenderness and muscle spasm are the rule in acute cases, while colitis with mucus and rectal bleeding is rarely seen since the organism is localized to the small intestine.

There have been reports of appendicitis and Meckel's diverticulitis due to obstruction of these intestinal appendages with parasites.[10]

Sensitization occurs as the organisms travel through the body, and eosinophilia and urticaria may be seen. Some experts think that asthma occurring with Ascaris infestation is a result of sensitization,[15] while others believe that wheezing is due to physical obstruction of the airways by the third-stage larva.

Diagnosis

Because the tools available for diagnosis vary from place to place, technique for diagnosis must also vary.

Definitive diagnosis. The definitive diagnosis may be made by identification of the worm in stool, vomitus, or sputum, or by identification of fertile or infertile ova in the stool.

A stool smear is prepared by suspending a drop of stool in a drop of normal saline on a clean glass slide. No stain is necessary for ova although some laboratory personnel use stain in order to bring out other ova or parasites that may be in the stool. A coverslip is placed over the suspension, and the specimen is examined under scanning lens and low power.

The infertile eggs vary in shape, some being bizarre, but most being elongate, about 90 by 40 micra. There is a thin hyaline shell with an irregular coat beneath that imparts a wavy appearance to the border of the egg. The protoplasm is coarsely granular.

Fertile eggs show more of a regularity in shape and closely approach a spherical appearance. They average about 60 by 40 micra, with an outer coat that is heavily lamellated and albuminoid. The shell is thick with a fatty inner coat. The protoplasm of young eggs contains lecithin granules which become finer as the egg embryonates.[15]

Presumptive diagnosis. In most areas microscopes are available so that a definitive diagnosis can be made. However, in some areas the incidence of Ascaris infestation in the stool is so high that we must look for other causes of symptoms despite the presence of Ascaris in the stool. When upper respiratory infection, acute surgical abdomen for other causes, hepatitis, and viral gastroenteritis can be ruled out, vague abdominal complaints can be taken for presumptive evidence of Ascaris infestation with or without a positive stool preparation when they occur in an endemic area. In the tropics and subtropics, eosinophilia as high as 40, 60, or 80 per cent of the white blood cells can be taken as presumptive evidence of parasitosis. In asymptomatic children living in highly endemic areas where 90 to 95 per cent of children harbor Ascaris, pallor and/or a protuberant abdomen may be taken as presumptive evidence of ascariasis.[16] When one sibling harbors Ascaris and where sewage systems are poor, a presumptive diagnosis of ascariasis in the other siblings may be made without laboratory or physical examination.

Suspicion. The disease should be suspected in any child in the tropics or subtropics and in any adult where hygiene or sewage disposal is poor. Any patient between 40° north and south latitude complaining of vague abdominal pain should be suspected of harboring Ascaris.

Symptoms referable to hepatic, pulmonary and cardiac systems are quite rare but when they occur in endemic areas should lead to a suspicion of ascariasis. Hepatomegaly, irregular liver edge, and upper quadrant tenderness should all be worked up with a differential white blood cell count and examination of stools for ova when they occur in endemic areas. Hemorrhagic pneumonia, wheezes, and chest pain are suggestive. X-ray demonstration of calcified granulomata in the lungs or liver or of irregular liver outline is suggestive of ascariasis or other parasitosis.

Sophisticated diagnosis. Rarely is it necessary to use techniques such as liver biopsy for diagnosis, but on several occasions this has revealed a cause of hepatic nodularity to be helminthic.[16] Persistently high eosinophil fraction of the white blood cell differential without evidence of worms may be an indication for bone marrow or small bowel biopsy. If these show elevated eosinophil production or deposition, benign eosinophilic leukemia or eosinophilic enteritis may be entertained.[16] An atypical pneumonia with eosinophilia is suggestive of tropical eosinophilia (Loeffler's syndrome), but long-bone evidence of granulomata is more suggestive of a histiocytosis when accompanied by atypical pneumonia and eosinophilia (eosinophilic granuloma).

More modern approaches to diagnosis are primarily autopsy procedures. Fluorescent antibody techniques have been of value in distinguishing Ascaris from other helminths in calcified pulmonary nodules.[17] Examination of emboli in coronary arteries of patients who died prematurely of acute myocardial infarction in endemic areas has revealed Ascaris ova in at least 1 case.[18] In addition, pathologic examination of the myocardium has revealed ascaridic granulomata.[18] In these patients arrhythmia and death may be the only symptoms. Autopsy examination of the nervous system may reveal an ascaridic cause for idiopathic encephalitis and/or meningitis.[15]

Diagnosis by skin-testing. There has been some progress made in the production of antibodies against Ascaris protein in rats.[19] Although this may have some use in the future in treatment of ascariasis, its use as a diagnostic skin test is in doubt. While it may be useful in temperate areas for the diagnosis of ascariasis as an obscure disease, the widespread prevalence of the antigen would make its use in more tropical zones virtually useless at the present time.

Treatment

There is some discussion as to whether asymptomatic carriers of Ascaris ova should be treated. Vines et al[7] found no differences in hemoglobin levels and nutritional status between asymptomatic carriers and noncarriers and suggest that treatment be reserved for those with symptoms. Because of the large number of complications in ascariasis all children in our clinic with Ascaris in the stool are treated with piperazine on a weight-related dose scale (table 3). A prime function of the pediatric clinic is to teach methods of hygiene and to aid developing countries as best it can in methods of sewage control and toilet facilities. The general feeling exists that as these improve and as treatment of the worm continues, the incidence of ascariasis will continue to fall.

TABLE 3: Dosage Table for Piperazine Given Orally in the Treatment of Ascariasis in Children*

Weight in Pounds	Dosage in Teaspoonfuls	Number of Administrations—Comments
A. Rare Ascaris ova seen in stool smear:		
Less than 40	2	1
40 to 60	4	1
60 to 100	6	1
More than 100	8	1
B. Few or occasional Ascaris ova seen in stool smear:		
Less than 40	2	2, 12 to 24 hrs. apart
40 to 60	4	2, 12 to 24 hrs. apart
60 to 100	6	2, 12 to 24 hrs. apart
More than 100	8	2, 12 to 24 hrs. apart
C. Many Ascaris ova seen in stool smear:		
Less than 40	2	2, 12 to 24 hrs. apart; repeat smear 1 week
40 to 60	4	2, 12 to 24 hrs. apart; repeat smear 1 week
60 to 100	6	2, 12 to 24 hrs. apart; repeat smear 1 week
More than 100	8	2, 12 to 24 hrs. apart; repeat smear 1 week

* As per Dr. J. Wershing, Agnes Hardecker Children's Clinic, Nassau, Bahamas.

If Ascaris is found in association with hookworm, Ascaris is treated first.

Piperazine. Piperazine was first used in the early 1900's in the treatment of gout because of the high solubility of uric acid in the compound. However, it was not long before it was shown to have little or no uricosuric value in vivo. Experiments with the drug showed its very low toxicity, and when its anthelmintic action was demonstrated by Boismare and later Fayard in 1949 it was widely accepted immediately.

The action of the drug is to paralyze the muscle coat of the worm, apparently by blockage of acetylcholine effect. The worm is then passed by peristalsis.

The drug is absorbed from the gastrointestinal tract and so is effective against all stages of the worm as it migrates throughout the body. Only a small amount of piperazine is broken down, the rest being excreted by the kidneys.

Although mild intestinal upset, urticaria, and transient neurologic changes have been reported, there is a very wide range between therapeutic and toxic doses. There are no contraindications to its use.

The common preparation is 100 mg. per ml. of piperazine citrate, U.S.P.[20] A single dose, as indicated in table 3, is given when there are rare ova seen in the stool. If worms have been seen by the patient, or if few or occasional ova are seen on stool smear, a second dose is given in 12 to 24 hours, and if many ova are seen, 2 doses are given and stool examination is repeated in 1 week.

Piperazine is almost universally effective against Ascaris. In fact, when many worms are present in the host, a massive bolus of worms paralyzed by piperazine may *en passage* cause intestinal obstruction. In some clinics it is found best to tranquilize the worms with reserpine before giving a paralyzing dose of piperazine, so that the worms are passed in a flaccid rather than spastic state, and hence are less likely to cause obstruction. We have never found this necessary in our experience. Reserpine alone has been effective as a vermifuge in veterinary medicine, and experiments with it as such in humans were our initial introduction to its antihypertensive and major tranquilizing qualities.

Hexylresorcinol. This drug is effective in 90 per cent of Ascaris infestations with 1 or 2 treatments. However, its high toxicity and the availability of piperazine have lessened its use. From its discovery in 1931 it was used as the drug of choice against ascariasis until 1949 when the anthelmintic property of piperazine was presented.[20] It is used now in cases resistant to piperazine therapy.[21]

Older drugs. Other drugs are available for the treatment of ascariasis, but their interest is primarily historical. Diethylcarbamazine must be given over a longer period of time, and is also more toxic. Its use lies in its effect against Wuchereria, Loa loa, Onchocerca, and Toxocara. Bephenium has some effect against Ascaris in combination with hookworm, but its use is primarily against the latter. Dithiazanine is very effective but also very toxic. Its prime use is against Litomosoides.[20]

Newer preparations. Many new preparations are being used abroad for the treatment of ascariasis, among them tetramisole[22] and thiabendazole.[23] The latter has important potential against *Trichinella spiralis*.[20] The availability and success of piperazine make research with these drugs in our country unrealistic.

Treatment of combined infestations. The value today of these alternative ascarifuges may be in their ability to combat combined infestations. However, it is our belief that Ascaris should be treated primarily and other infestations secondarily. In the case of pinworm this helminth will usually respond to the piperazine therapy for Ascaris,[20] although combinations of piperazine and Povan® (pyrvinium pamoate) are available.[24] If piperazine alone fails, Povan should be given independently. Of the parasites* commonly encountered in our clinic only *Entamoeba histolytica* infestation receives priority over Ascaris in treatment.

**Trichuris trichiura, Giardia lamblia, Chylomastix mesneli*, trichomonads, and *Entamoeba histolytica*.

Summary

Ascaris lumbricoides is a nematode with a very high predilection for the human intestine. Its incidence is highest where poor sewage and poor hygiene combine with a tropical climate amenable to the development of the Ascaris ovum. It infests 35 to 94 per cent of children in tropical areas. Infestation is complicated by intestinal, hepatic, pulmonary, cardiac, and neurologic symptoms.

Experience with Ascaris at the Agnes Hardecker Clinic in the Bahamas has been presented. A clinic in the tropics has among its first concerns education of the developing natives in terms of sewage control and hygiene. Vermifuge itself is with piperazine citrate in a weight-related dosage schedule.

The classification, life cycle, and anatomy of *Ascaris lumbricoides* have been given. The recent literature has been reviewed with regard to complications and newer methods of therapy.

(*Continued on page 413*)

The Hazards of High Parity*

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NOTWITHSTANDING THE EFFORTS to limit population in certain areas of the world, there are areas where, due to custom, religion, or national policy, the very opposite reproductive practices prevail. This must be reckoned with as an obstetrical fact of life, all aspects of which bear examination. This is especially true in view of the fact that high parity has been equated with high risk.[1, 2, 3] This report attempts to assess the risks.

Material and Method

Among a total of 5,579 patients delivered during 5 years (1963–1967), there were 230 patients ranging in parity from 10 to 16. This high parity group was reviewed in order to determine the relationship of parity to obstetrical and related complications. Additional observations were made pertaining to the newborn in relation to high parity, an analysis of the factor of patient-origin, and reproductive practices (table 1).

TABLE 1: Incidence of High Parity

	Total Cases—General Obstetrics Series	Total Cases—High-parity Series	Per cent	Parity
Zefat General Hospital	5,579	230	4.6	10 or over
Israel and Blazar[1]	128,568	5,529	4.3	7 or over
Muth and Legerlotz[2]	50,086	1,853	3.7	6 or over
Rouchy and Grosieux[3]	5,745	339	5.9	8 or over

Results

Origin and reproductive practices. Over 95 per cent of the patients were born in countries bordering on the Mediterranean or in the Near and Middle East. In these patients, regardless of racial origin, a high parity is highly regarded on both social and religious grounds.[4] It is a distinction of virility and a sign of affection for the woman, and is frequently indispensable to a successful marriage. Sterilization, even on purely medical grounds, is resisted. Contraceptive measures have made no serious inroads. A hysterectomy for prolapse or other gynecologic lesion in a Para 10 may be considered a tragic loss. According to religious proscription coitus is restricted to specified periods.§ Marriages were entered into at the age of 12 and younger.|| Sterility after 10 years of cohabitation is grounds for divorce.§ The overwhelming predominance of a common racial stock and similar demographic origin are factors which must be considered in evaluating the results.

Age. The patients ranged in age from 25 to 48 years (table 2). Almost 50 per cent were between the ages of 35 and 39, 71 per cent were under 40, and 29 per cent were between the ages of 40 and 48. The incidence of complications in the oldest group (40 and over) is higher. Although it represents only 29 per cent of the cases, it shows an incidence of 30 per cent of the complications. The majority of the postpartum hemorrhages requiring transfusions occurred in this group.

TABLE 2: Relative Distribution of Parity and Age

Age	Parity							Total	Per cent
	10	11	12	13	14	15	16		
Under 35	23	17	7	2	3	1	—	53	23
35–39	36	32	26	8	6	2	—	110	48
40 and over	30	13	6	7	7	2	2	67	29
Total	89	62	39	17	16	5	2	230	100
Per cent	38.7	26.9	17.0	17.4					

Parity. The incidence of 4.6 per cent in our series closely approximates the figures of Israel and Blazar,[1] 4.3 per cent, and of Muth and Legerlotz,[2] 3.7 per cent. The complications did not appear to vary significantly in relation to parity, either in incidence or severity. The incidence of complications ranged in descending order of frequency as follows: Para 10 (38.7 per cent) had 42.6

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§Refers to patients of the Hebrew faith, the predominant group, and specifies that coitus may be practiced only after a ritual bath (*mikva*) taken on the fourteenth day of the cycle, and provided all signs of menstruation have disappeared.

||Since the establishment of the state of Israel marriage under the age of 17 is prohibited by law.

per cent of the complications. Para 11 (26.9 per cent) had 20.6 per cent of the complications. Para 12, with 17 per cent of the patients, had 25 per cent, while the remaining groups comprising 17.4 per cent accounted for only 11.8 per cent of the complications.

Abortions. The total number of abortions which occurred during the reproductive life of this group of patients was 196 (8.1 per cent). This was approximately one-third of the general obstetrical abortion rate of 25.7 per cent. There was no relationship noted in the frequency of abortions in relation to age.

Perinatal deaths. A progressive increase was observed also in the number of perinatal deaths with rise in parity. This incidence rose sharply with parity 13 and over.

Sex distribution of babies. Whereas the reported sex distribution of male to female births is 104 to 100,[5] the distribution in our high parity series was 124 males to 100 females.

Maternal mortality. While the general maternal mortality for the hospital is 12 per 10,000 live births, no maternal deaths occurred in this series.

Perinatal mortality. This was 4.7 per cent. It is somewhat higher than the 3 per cent perinatal mortality for the general hospital series. Of these fetal deaths 45 per cent were attributed to prematurity. The remainder died of respiratory distress syndrome and atelectasis.

Complications

All of the complications which occurred in the high parity series are listed in table 3 and are compared with percentage incidence of the general obstetrical hospital series. On the whole, there is no marked departure from the incidence in the general series. In some aspects, however, there appears to be a considerable increase, notably in premature rupture of the membranes, cord prolapse, abruptio placentae, secondary inertia, and breech presentation. A slight increase is noted in the incidence of postpartum hemorrhage, retained placenta, and postpartum sepsis. On the other hand, the indications for operative delivery by forceps and vacuum extraction are diminished. The frequency of delivery by cesarean section is somewhat less. There is a significant reduction in the incidence of complications due to preëclampsia, chronic nephritis, diabetes, and rheumatic valvular disease.

Discussion

While it may appear as a discordant note to discuss the subject of high parity at a time of "population control," nevertheless 3 to 4 per cent of patients will continue to qualify in the foreseeable future. Evaluation of results in this series must take into consideration that the patients who comprise it come from a very low economic sector. These women labor hard in the homes and in the fields of an agricultural mountain community. Opportunities for self-indulgence are few, and malnutrition and nutritional anemia are common because "there are so many mouths to feed." Life expectancy by Western standards is low. Furthermore, the cases were selected on a higher parity basis than generally reported.

TABLE 3: Comparative Percentage Incidence of Complications in High-parity and General Hospital Series

Complication	High Parity Series (per cent)	General Hospital Series (per cent)
Early rupture of membranes	4.50	2.80
Breech presentation	6.80	4.30
Prolapse of cord	1.60	0.60
Retention of placenta or cotyledon	3.60	2.50
Postpartum hemorrhage	4.30	3.80
Transfusions	4.70	4.30
Sepsis	3.90	2.40
Forceps and vacuum extraction	2.60	3.50
Cesarean section	1.70	2.10
Uterine atony	0.43	1.30
Primary inertia	0.43	0.33
Secondary inertia	2.10	0.33
Abruptio placentae	1.30	0.35
Placenta previa	0.43	1.40
Abortion	8.10	25.70
Prematurity	1.70	4.70
Preëclampsia	0.86	3.50
Chronic nephritis	0.08	0.30
Diabetes	0.04	0.10
Rheumatic carditis	0.04	0.30

It should also be noted that 29 per cent of the patients were between the ages of 40 and 48. Notwithstanding all this, there was no maternal mortality. Where an increase in complications was noted over that of the hospital series in general, it was not so significantly higher as to reveal high parity of this bracket to be a prohibitive obstetrical risk. The quantitative increase in complications, where it did occur, did not compare in magnitude with other reports.[6, 7, 8] Certainly we found no justification in high parity per se for immediate sterilization after delivery of the eighth child, as advocated by some.[9] Since with advancing parity there is a parallel advance in age, it really becomes difficult to separate the effects of parity from those of age.[10, 11] From the standpoint of reproduction, the high parity patient has demonstrated superlative endowment, and therefore should be, in a way, expected to produce better results than the "common run" of the hospital series. Efforts to attribute increased bleeding complications, inertia, atony, and disruption of the uterus to a diminishing elastic tissue component[2] with advancing parity have been disproven by others.[12] Moreover, the very high-parity patient is more fortunate than her less experienced sister of lower parity who may still be a candidate for diabetes, chronic nephritis, essential hypertension, or other medical ravages. Our very high-parity patient has demonstrated a lower incidence of these complications, otherwise she might not have reached this high-parity level.

Local anesthesia was employed in the vast majority of these patients. Where there is uterine fatigue with a tendency to atonia, the role of general anesthesia as a precipitating factor in hemorrhage is worth considering.[12]

Finally, generally improved obstetric practices and availability of antibiotics, oxytocics, and blood all undoubtedly contributed to a reduction of complication or minimization of their effects.

Conclusions

The frequency of complications in a group of Para 10 to Para 16 patients was only slightly higher than in the general hospital series.

The complications which characteristically occur in high-parity women involving diminished uterine tone and contractility resulting in inertia and third stage problems are readily manageable with modern hospital facilities.

A higher than average incidence of male births was noted in the high parity series.

Abortions occurred one-third as frequently in the high parity series as in the general hospital series, which suggests a more adequate reproductive ability in these patients.

The high-parity patient should be considered an obstetrical challenge but not a prohibitive risk.

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Ascariasis—Greenbaum

(*Continued from page 410*)

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Venomous Snakebites Treated in the District of Columbia

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While patients referred for treatment of venomous snakebites are rare in the District of Columbia, bites by these snakes in the District of Columbia are even less common. Most snakebite patients treated by physicians and in hospitals in the District of Columbia received their bites in nearby Virginia and Maryland. The District of Columbia is listed along with several states as comprising the South Atlantic Region of the United States. The annual rate of treated venomous snakebites per 100,000 population in the political units of this region are: North Carolina—18.79, Georgia—13.44, West Virginia—11.29, South Carolina—7.72, Florida—7.07, Virginia—5.47, Delaware—1.79, Maryland—1.35, and the District of Columbia—0.65.[1] There were no fatalities resulting from snakebites in the District of Columbia during the 10-year period, 1950 through 1959.[2]

Venomous Snakes

The only venomous snake currently indigenous to the District of Columbia and vicinity is the northern copperhead, *Ancistrodon contortrix mokeson*. According to Klauber,[3] although the timber rattlesnake, *Crotalus horridus horridus*, once existed in the District of Columbia, it seems now to have been exterminated. However, an occasional specimen may still be carried in by a river flood.

The copperhead is so named because of its coppery-red head. Also, it is commonly called the dryland moccasin, highland moccasin, chunkhead, pilot snake, poplar-leaf snake, red-oak snake, and white-oak snake. The average adult copperhead is 60 to 90 cm. (24–36 inches) long. It has a large, coppery-red head, a moderately thick body, reddish-brown hourglass markings on the sides of its body, and a tail ending in a point, without rattles (figure 1). A young copperhead's tail may have a yellow tip. This terrestrial snake's favorite haunts include mountains, wooded hillsides, rock piles, rock quarries, and sawdust piles. It is not unusual for copperheads to live within city limits and near suburban housing developments. Mice, other rodents, birds, insects, and frogs constitute their major source of food. Copperheads are found in 30 states in the eastern two-thirds of the United States.

How can one distinguish a copperhead from a harmless snake? Copperheads are pit vipers. They are identified by a characteristic pit between the eye and the nostril on each side of the head, by elliptical pupils, and by 2 well-developed fangs which protrude from the maxilla when the mouth is opened. By turning the belly upwards one may observe a single row of plates under the tail except near the tip. Harmless snakes do not have facial pits, have round pupils and small teeth but no fangs. The underside of a harmless snake's belly shows a double row of subcaudal plates.

Material and Method

The data for this report were obtained from a national survey of venomous snakebites.[1] A questionnaire and letter were mailed to a group of District of Columbia hospitals listed in the American Hospital Association's Guide. We selected general hospitals, children's hospitals, college infirmaries and Army, Navy, Coast Guard, Public Health Service, Air Force, and Veterans Administration hospitals. Maternity, tuberculosis, and mental hospitals were omitted. A total of 17 District of Columbia hospitals comprises the study group. Each hospital was requested to report all inpatients admitted for snakebite during 1958 and 1959.

Most hospitals do not code and tabulate the diagnoses of emergency room and outpatient clinic visits. Since some snakebite victims are not admitted to the hospital as inpatients, it seemed essential to ask a sample of practicing physicians how many snakebite victims they treated on both an outpatient (office, home, emergency room) and on an inpatient basis. Previous surveys have shown that most people with venomous snakebites are treated by general practitioners, surgeons, internists, pediatricians, and orthopedic surgeons. A random sample of one-third of all the District of Columbia physicians in these categories of practice who were listed in the American Medical Directory were sent questionnaires.

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Death certificates for fatal snakebite cases were obtained from the District of Columbia Department of Public Health.

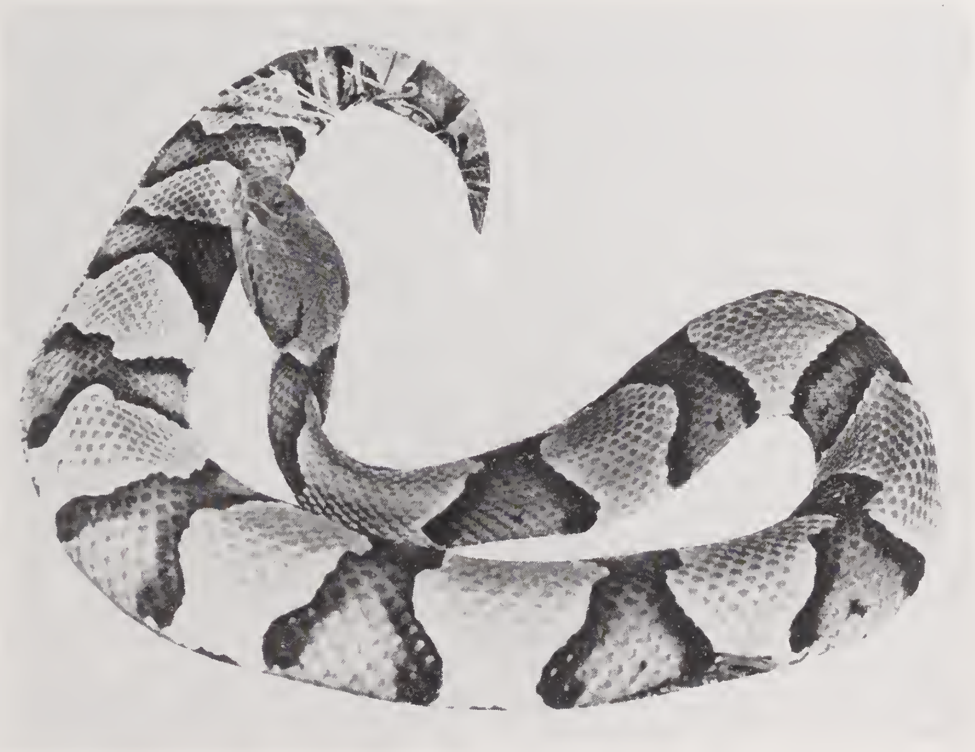

FIG. 1. Copperhead.

Results

This report is based on questionnaires returned by all 17 District of Columbia hospitals. It is supplemented by questionnaires returned by 223 (76 per cent) of 293 practicing physicians. The District of Columbia Department of Public Health reported that there were no snakebite deaths during 1958 and 1959.

Hospitals reported a total of 4 inpatients treated during 1958 and 1959. Physician reports (adjusted to account for all District of Columbia physicians in the relevant practice categories) indicated that 3 inpatients and 2 outpatients were treated for snakebite accidents each year. Thus, approximately 5 people are treated annually for venomous snakebites in the District of Columbia. This provides an incidence of 0.65 bites per 100,000 population per year. Of interest is the fact that only 1 bite accident happened in the District of Columbia. The remaining patients were referred to Washington hospitals for treatment after they were bitten in nearby Virginia and Maryland. Virginia ranks fifth and Maryland ranks seventeenth in the annual incidence of treated copperhead bites in the United States.[4]

Of snakebite victims treated in the District of Columbia, 4 were bitten by copperheads and 1 by an unidentified venomous snake (most likely a copperhead). There were too few copperhead bites in the District of Columbia for a meaningful epidemiologic analysis. However, the following facts about copperhead bites were obtained from a nationwide survey.[4] Males have higher bite rates than females, and persons in the 10- to 19-year age group have relatively high bite rates. Whites have higher bite rates than nonwhites. Most copperhead bites occur from April through October, with peak months in July and August. About 43 per cent of copperhead bites happened in the victims' own yards. An additional 12 per cent were in, under, or on the porch or patio of a building. Ninety-eight per cent of the bites are inflicted on the extremities, 36 per cent on the upper and 62 per cent on the lower.

Severity of Venenation

The severity of venenation from copperhead bites may be determined by a modification of the classification of Wood et al.[5] Bites are classified as follows:

Grade O, no venenation. Fang or tooth marks, minimal pain, less than 1 inch of surrounding edema and erythema. No systemic involvement.

Grade I, minimal venenation. Fang or tooth marks, severe pain, 1 to 5 inches of surrounding edema and erythema in first 12 hours after bite. Systemic involvement usually not present.

Grade II, moderate venenation. Fang or tooth marks, severe pain, 6 to 12 inches of surrounding edema and erythema in first 12 hours after bite. Systemic involvement may be present—nausea, vomiting, giddiness, shock, neurotoxic symptoms.

Grade III, severe venenation. Fang or tooth marks, severe pain, more than 12 inches of surrounding edema and erythema in first 12 hours after bite, systemic involvement usually present as in grade II.

Of 701 copperhead bites in the national survey with detailed clinical information available, 20 per cent were grade 0, 42 per cent were grade I, 27 per cent were grade II, and 11 per cent were grade III. It is important to recognize that not every bite by a venomous snake results in signs and symptoms of venom poisoning. This paradoxical phenomenon may result from intervening clothing on the victim, from indirect hits with the fangs, from a lack of venom in the snake's venom glands owing to recent feeding, or from an unexplained failure of the fang-venom gland mechanism. With adequate medical treatment there should be few fatalities from copperhead bites. However, severe copperhead bites that go untreated may result in death of the patient. Delayed and/or inadequate treatment accounts for most snakebite fatalities in this country.[2]

Treatment

The following treatment currently is recommended for copperhead bites. A constricting band (tourniquet) should be applied lightly to the involved extremity several inches above the bite. It should be applied only tight enough to occlude the superficial venous and lymphatic flow. It should not occlude the arterial circulation, and it should be released every 10 to 15 minutes for a minute or 2. As edema resulting from venom poisoning spreads, the constricting band should be advanced to keep just ahead of the swelling. The purpose of the band is to impede the spread of venom until incision and suction can be used to remove the venom mechanically or until polyvalent crotaline antivenin can be administered to neutralize the venom.

Incision and suction are effective in removing venom from experimental animals up to about 60 minutes after the venom is injected. The sooner it is used, the larger the amount of venom that can be removed. Suction should be used for about 1 hour. Incisions, $\frac{1}{4}$ inch long and $\frac{1}{8}$ to $\frac{1}{4}$ inch deep, are made into the subcutaneous tissues over the fang punctures.

The "3 A's" (antivenin, antibiotics, and antitetanus prophylaxis) are recommended in addition to incision and suction in the treatment of all serious pit-viper bites. Antivenin manufactured in this country is effective in neutralizing the venoms of all North American pit vipers. Since antivenin is produced from horse serum, the patient should receive a skin test before antivenin is given. Antivenin diluted in 500 ml. of normal saline may be given intravenously. Studies with radioisotopes have shown that antivenin accumulates at the site of the bite more rapidly after intravenous administration than after intramuscular administration.[6] Although antivenin has been administered intravenously for several decades in other countries, this route of administration was not used frequently in the United States before 1960. Injection of antivenin into the local bite area is not a particularly effective way to administer antivenin. We have found the following amounts of antivenin useful in treating the various grades of venenation: grade O (no venenation) requires no antivenin, grade I (minimal venenation) may require 10 ml. (1 ampule) of antivenin, grade II (moderate venenation) requires 30 to 40 ml. of antivenin, and grade III (severe venenation) requires 50 ml. or more of antivenin. When large doses of antivenin are administered one should anticipate that serum sickness will develop as a complication of treatment in a high proportion of patients.

Since snakes' mouths and venoms may harbor pathogenic organisms, antibiotics and tetanus toxoid or tetanus immune globulin should be given prophylactically. Since gram-negative organisms predominate, a broad-spectrum antibiotic is indicated.

Corticosteroids and corticotropin (ACTH) do not affect the survival rate of animals poisoned with pit-viper venoms.[7] They probably should not be used during the first few days after venenation, since they may interfere with venom-antivenin reaction. They may be beneficial later in treating serum sickness resulting from antivenin therapy. Antihistamines are contraindicated because they shorten the survival time of animals poisoned with pit-viper venoms. Shock resulting from venom poisoning should be treated with infusions of blood, plasma, saline solution, and vasopressor drugs. Analgesics are needed to relieve pain. Recently there have been reports of excessive tissue necrosis and amputations associated with cold therapy such as packing an extremity in ice or ice water or using ethyl chloride.[8, 9] In my opinion, cold therapy should not be used.

Summary

The northern copperhead, *Ancistrodon contortrix mokeson*, currently is the only venomous snake indigenous to the District of Columbia.

From a survey of physician reports, hospital records, and death certificates it was estimated that approximately 5 people are treated annually for copperhead bites in the District of Columbia. Most of the victims were bitten in nearby Virginia and Maryland and were referred to Washington for treatment. There were no snakebite fatalities.

The currently recommended treatment for copperhead bites includes incision and suction, the "3 A's" (antivenin, antibiotics, and antitetanus prophylaxis), and general supportive measures.

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Ways to Reduce Boating Accidents

To reduce the number of boating accidents, the American Red Cross offers these safeguards that boat operators and passengers should observe:

1. Carry a Coast Guard-approved life jacket for everyone aboard. Insist that weak swimmers and nonswimmers wear them at all times, and that others wear them in hazardous weather conditions.

2. Don't overload the craft. Keep passengers down to a safe number.

3. Don't overpower your boat. Consult your dealer on what horsepower motor it can take.

4. Don't smoke while refueling.

5. Don't let passengers in a small boat stand up or sit on the gunwales while you are under way.

6. Equip your boat with essential gear—anchor, oars, boat hook, extra line, fire extinguisher, tool kit, and first aid kit. Running lights are a must for night-time operation.

7. Check weather bureau forecasts before taking your boat out on open water. Take a transistor radio along to keep posted on changing weather conditions.

8. Most boats, if swamped or capsized, will stay on the surface, so stay with the boat when such accidents happen until help arrives. But if you are in dangerous or extremely cold water, try to get to shore as fast as possible.

Following are the basic rules of the waterways:

1. When boats are meeting in, or nearly in, a head-on position, each should keep to the right.

2. When boats are in a crossing position, the one on the right has the right of way.

3. When one boat is overtaking another, the boat being overtaken has the right of way.

4. Power boats should give way to manually propelled or sail boats unless the sail boat is overtaking a power boat. Small sail boats, when sailing in a channel, should give way to power boats which cannot operate outside the channel.

5. Sail boats to windward must keep out of the way of sail boats to leeward.

When a weak or nonswimmer falls overboard, swing the stern away from the side from which he fell. Toss him a life-saving device and promptly maneuver the boat back to him, making the approach as you would to a mooring—at reduced speed and slightly to windward if possible. Shut off the motor within a few feet of the victim and bring him aboard.

Angiographic Study of Carcinoma of the Pancreas

Report of a Case

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In spite of great advancement in radiology in the past years the diagnosis of carcinoma of the pancreas remains one of the most difficult problems among radiologists. Conventional radiologic procedures have no way to opacify the parenchyma of the pancreas. Roentgen diagnosis is limited chiefly to indirect signs of pancreatic disease. These are almost exclusively related to the effect of an enlarging pancreas on its neighboring structures. It is difficult to distinguish between a carcinoma of the pancreas, a benign process, and normal variations.

Since the introduction of the percutaneous method of arteriography,[1] selective celiac and superior mesenteric arteriograms can be easily and safely made and have been utilized for the diagnosis of carcinoma of the pancreas by many authors.[2-13] Although the experience is still limited, it appears to be the most valuable method for precise diagnosis of carcinoma of the pancreas. Recently we studied a case of carcinoma of the pancreas in this way.

Report of Case

R. H. (G.W.U.H. No. 3-66727), a 61-year-old Negro, was admitted to the George Washington University Hospital on February 20, 1968. One week before he had observed light-colored stools and dark urine. He had noted slight distention of the abdomen for 1 year but had generally been in good health. For 10 days prior to admission he had anorexia and experienced a 14-pound weight loss. In addition, for several days he had a sensation of food "sticking" in the upper abdomen. Five days prior to admission his private physician noted jaundice. There was no past history of nausea, vomiting, fever, weakness, diarrhea, constipation, or abdominal pain. There was no history of exposure to persons with hepatitis or jaundice, and the man had not ingested clams or oysters or well water. He had not received recent injections or transfusions and had not been taking any medications or received anesthesia. He had a history of drinking 1 pint of whiskey daily for a number of years. In 1932 he had a similar episode which was treated by his private medical doctor with "pills" and was well in 1 week.

The patient was well-developed, well-nourished, and in no acute distress. The blood pressure was 150/90, the pulse rate 88, and the respiratory rate 16. The positive physical findings were cutaneous, scleral and mucous membrane icterus and a positive spleen percussion sign but not a palpable spleen.

Laboratory data: urinalysis normal, hemoglobin 14.2 Gm. with a hematocrit of 44 per cent, white blood cell count 4,400 with a normal differential, fasting blood sugar 84 mg., blood urea nitrogen 11 mg., prothrombin time 14 and 12.5 seconds, serum sodium 141 mEq./L, serum CO_2 22 mEq., serum chloride 105 mEq., serum amylase 75 units, serum lipase 0.6 ml., thymol turbidity 2.6 and 3.6 units, serum calcium 10 mg., serum phosphorus 3.6 mg., cephalin flocculation test negative, bromsulfalein test for renal function 48 per cent retention, Kunkle globulins 7 and 4.7 per cent, serum lactic dehydrogenase 380, 330, 545, 445, and 465 units on different days, D-xylose 4.8 Gm. per 875 c.c. Serum protein electrophoresis showed albumin of 3.3 Gm. and gamma globulin of 1.6 Gm. Urine urobilinogen and bile were positive, the former in a 1–80 dilution. Stools were positive for urobilinogen. See other data (table 1).

Chest X-ray films and flat plate of the abdomen were not remarkable. Upper gastrointestinal X-ray examination showed no conclusive evidence of abnormality. A barium enema X-ray examination was within normal limits. An electrocardiogram showed left ventricular hypertrophy. A pancreatic scan was done, but the pancreas was not seen. A liver scan showed no enlargement of the liver.

On February 27, 1968 selective percutaneous celiac and superior mesenteric arteriography was performed. The celiac arteriogram revealed displacement of the hepatic artery superiorly and of the gastroduodenal artery toward the left (figure 1). The venous phase of the celiac injection failed to show the splenic vein, but the portal was seen through a collateral vein (figure 2). This indicated obstruction of the splenic vein. The

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TABLE 1: Significant Laboratory Data

	2-20	2-23	2-26	2-27	2-28	3-1	3-4	3-12	3-16
Cholesterol	306				262				
Bilirubin—direct/indirect	3.5/1.5	2.3/1.5	3.6/1.4	3.3/0.5	3/0.8		3/2.4	1.5/1.0	0.9/0.3
Total bilirubin	5	3.8	4.2	3.8	3.8		5.4	2.5	1.2
Alkaline phosphatase	10.5	12.5	13.2	13.4	12	15.5	13.4	8	11.7
SGOT	166	118	100	100	104	100	76		
SEGT	510	400	260	260		83	81	36	

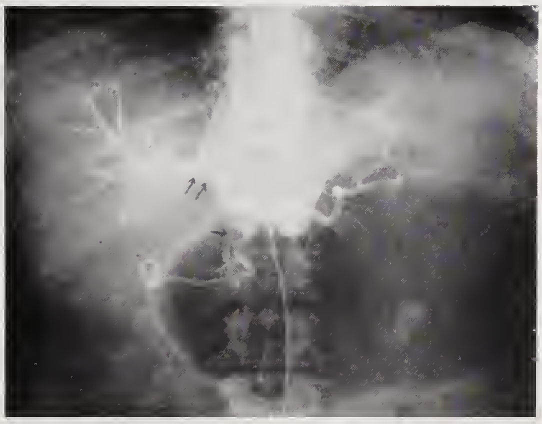

FIG. 1. Arterial phase of the selective celiac arteriography showed displacement of the hepatic artery (double arrow) and the gastroduodenal artery (single arrow).

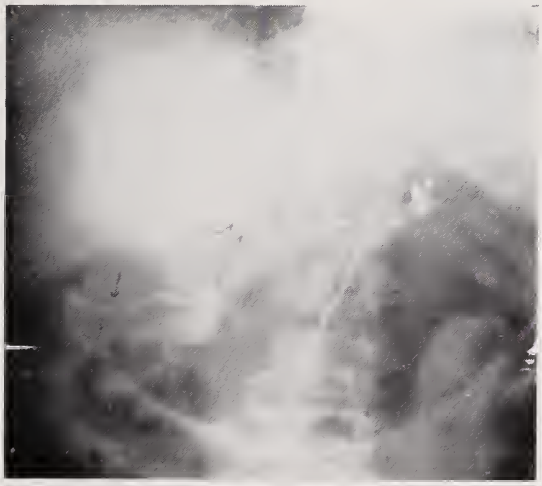

FIG. 2. Venous phase of the selective celiac arteriogram showed visualization of the portal vein (double arrow) through a collateral vein (single arrow).

spleen and the liver were not enlarged. The superior mesenteric arteriogram revealed questionable irregularity of vessels of the pancreatic arcades (figure 3). The venous phase of the superior mesenteric artery injection revealed extrinsic compression at the junction of the superior mesenteric vein and the portal vein (figure 4). Because of both arterial and venous changes the findings were highly suggestive of carcinoma of the pancreas.

On March 7, 1968 an exploratory laparotomy was performed and a biopsy of the pancreas taken which showed adenocarcinoma with neural invasion. Both the portal vein and the splenic vein were invaded by the carcinoma of the pancreas. A "by-pass" cholecystojejunostomy was performed.

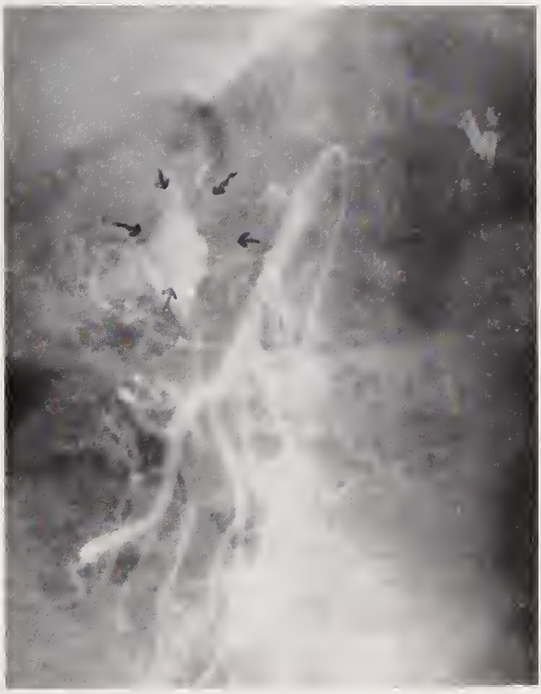

FIG. 3. Arterial phase of the selective superior mesenteric arteriography showed questionable irregularity of vessels of pancreatic arcades (between arrows).

Comment

Carcinoma of the pancreas in general lacks detectable pathologic tumor vessels, and only rarely can a tumor stain be appreciated.[3, 7, 9, 11, 12] Carcinoma of the pancreas may produce irregularity, truncation and disruption of small vessels of the pancreatic arcades which are difficult to detect. However, because of close anatomic relation between the pancreas and its adjacent vessels, namely, the celiac axis, splenic artery, hepatic artery, gastroduodenal artery, superior mesenteric artery, splenic vein, portal vein and superior mesenteric vein, and because of invasive growth of carcinoma of the pancreas, narrowing, occlusion and displacement of these adjacent vessels can be demonstrated by means of selective arteriography. Combined arterial and venous abnormalities located in the same region appear to be a reliable sign of carcinoma of the pancreas.[9]

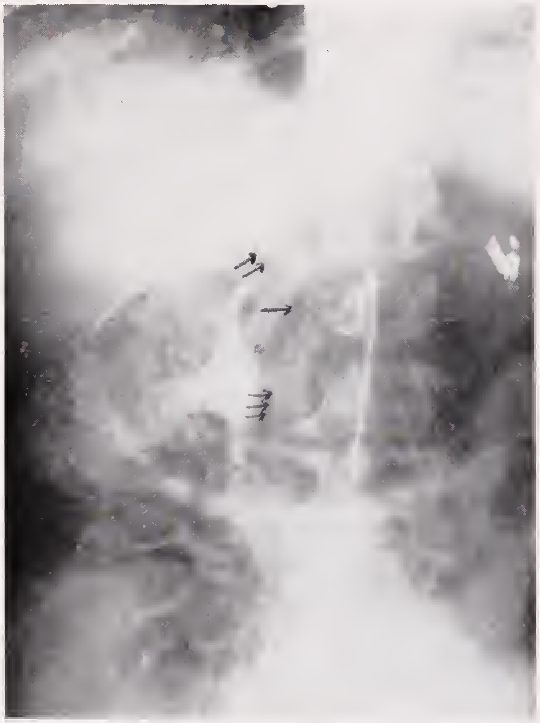

FIG. 4. Venous phase of the selective superior mesenteric arteriography showed extrinsic compression (single arrow) at the junction of the portal vein (2 arrows) and the superior mesenteric vein (3 arrows).

Summary

A case of carcinoma of the pancreas in which the laboratory and physical findings were inconclusive was studied by selective celiac and superior mesenteric arteriography, and a more definitive diagnosis was made. A brief discussion of the angiographic findings of carcinoma of the pancreas has been presented. Selective celiac and superior mesenteric arteriography appears to be one of the most useful radiologic procedures for the diagnosis of carcinoma of the pancreas.

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Memorandum on Recording Date on International Certificate of Vaccination

The World Health Organization has recently called attention to the requirement in the International Sanitary Regulations that all dates should be recorded in the following sequence:

DAY	MONTH	YEAR
e.g. 2	May	1968

WHO points out that difficulties continue to arise because of the use of arabic figures for recording the month. In the United States and some other countries, it is the custom to write the month (either in letters or arabic numerals) before the day. However, it is a common practice in many other countries to follow the format indicated above. Thus, an American physician who vaccinated a patient on November 2, 1965, may have written the date as "11/2/65." A European Quarantine Inspector would assume that such a certificate had been issued on February 11, 1965. He would, therefore, consider the certificate invalid because of issuance more than 3 years prior to the present time. No misinterpretation is possible, however, if the data were indicated as prescribed by WHO, that is, "2 Nov. 65."

James W. Mosley, M.D.
Chief, Epidemiology and Research Analysis Section, Foreign Quarantine Program, National Communicable Disease Center

Shigellosis in the District of Columbia

1967 Year-end Summary

CHARLES R. HAYMAN, M.D., M.P.H.*

During the calendar year 1967 there were 185 reports of human Shigella isolates received by the Epidemiological Services Division of the D. C. Department of Public Health. This compares with 69 reports during 1966 and a 10-year mean of 105. This represents the highest yearly incidence, there being 139 reports in the next highest year, 1961. Figure 1 shows the number of reports per month for the 10-year period, 1958-1967. The striking features are the seasonal variation, with peak incidence in the fall and early winter months, and the cyclic nature of the annual incidence. The seasonal variation corresponds quite closely with that observed nationally. The sharp increase in incidence this year can be accounted for by the unusually large number of cases in November and December. Data are unavailable for other cities, but of all U. S. jurisdictions reporting directly to National Communicable Disease Center, Washington ranks ninth in the second and third quarters of 1967 in Shigella incidence.

The breakdown of Shigella isolates this year as to serotype is as follows: Type D (*S. sonnei*)—136, Type B (*S. flexneri*)—24, Type A (*S. Dysenteriae*)—2, and unknown type—23, with a total of 185. This corresponds with the pattern in other areas of the northern United States.

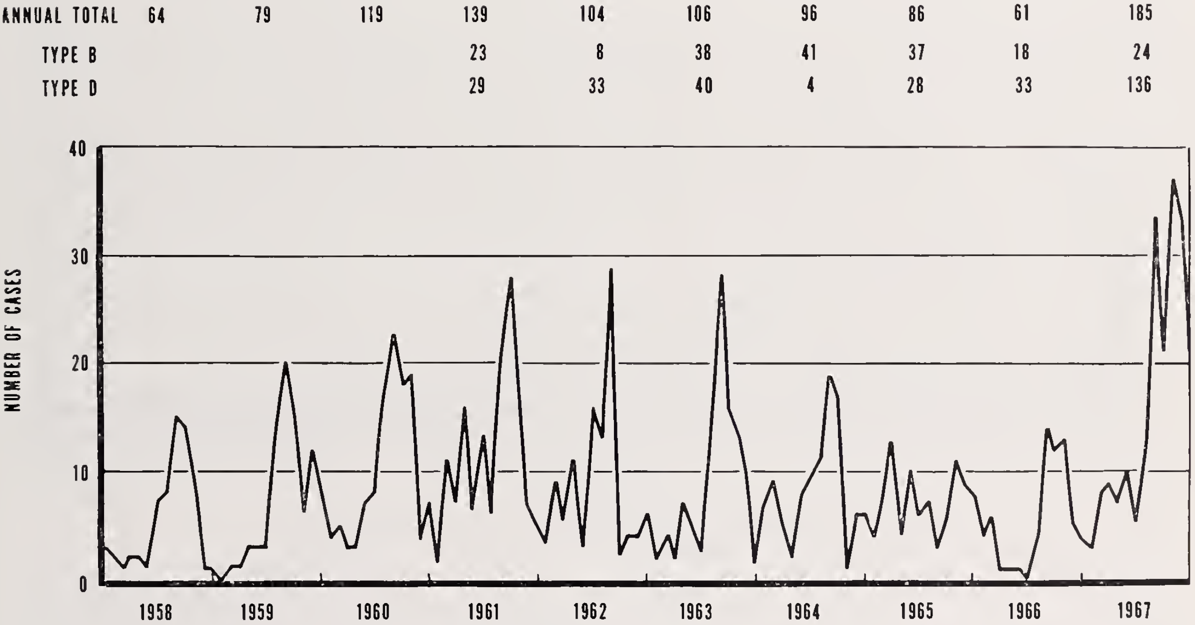

FIG. 1. Number of cases of Shigella reported per month for 1958–1967.

The age distribution of Shigella cases is shown in figure 2. All but 2 cases occurred in children. Of the 2 adult cases, one was the mother of an infected child and the other acquired Shigella while in Trinidad. Eighty per cent of cases were in preschool children (age less than 6 years). Maximum number of cases occurred at age 2 years. Of 185 cases, 167 were in Negro children.

The major concentrations occurred in the lower socioeconomic areas. Of the 185 cases, 37 were associated with poor sanitary conditions in the home. This consisted of overcrowding, general uncleanliness, and the presence of "roaches, rats and/or vermin." There were no instances of contamination of water supply by sewage or other plumbing irregularities. There was no suggestion from food histories of a possible common source outbreak, except for the 1 case acquired in Trinidad. None of the Shigella patients had contact with any of the others, except for 13 instances of positive family contacts involving 32 persons. Of the 292 family contacts cultured by the Health Department 19 were found to be Shigella carriers (6.5 per cent). The carrier rate for contacts 6 years of age or less was 10 per cent and for those greater than 6 years of age it was 5 per cent. Of the 185 patients, 49 required hospitalization.

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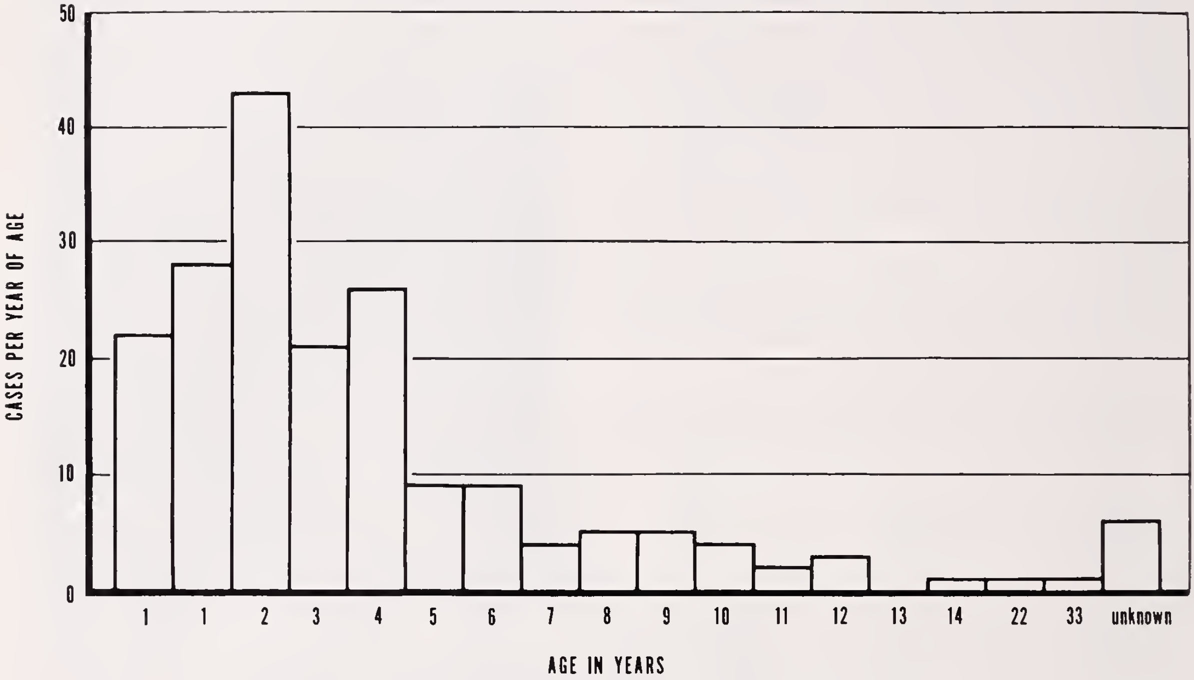

FIG. 2. Age distribution of Shigella patients for 1967.

The sources for reports of Shigella during 1967 are shown below.

Source	No. Reports
D. C. General Hospital	68
Children's Hospital	97
Freedmen's Hospital	5
Georgetown Hospital	2
Group Health Medical Center	2
Junior Village	1
Private physicians	1
Walter Reed Army Hospital	2
Contact of known case (D. C. Laboratory)	7
Total Cases	185

Reporting from D. C. General Hospital is complete and culture results are checked at the Health Department Central Laboratory. Reporting from Children's Hospital is probably complete. The extent of reporting of Shigella isolates from other D. C. hospitals is not known. The lack of reports from private physicians probably reflects the low incidence of Shigella in higher socioeconomic groups but may also indicate poor reporting, or treatment of patients having diarrhea without bacteriologic diagnosis.

Discussion

The epidemiologic pattern in the District of Columbia is that of endemic shigellosis, found in lower socioeconomic groups and consistent with a fecal-oral route of transmission. The large increase during 1967 cannot be explained by improved reporting or increased utilization of bacterial stool cultures for diagnosis of patients with diarrhea. Since the vast majority of reports come from 2 institutions where stool cultures have been taken routinely on such patients for a number of years, the increase in isolates probably reflects a real increase in infections. There has been no evidence this year of shigellosis at Junior Village. Similarly, there has been no evidence of epidemic shigellosis, i.e., that associated with a common source outbreak. The increase in reports during the last 2 months of 1967 reflects the unusually large number of *S. sonnei* isolates during this period.

Although the number of cases was unusually large, no concentration of cases could be found

(Continued on page 452)

Dermatofibrosarcoma Protuberans

Report of a Case*

GEORGE RIUS, M.D,† AND BRUCE I. SHNIDER, M.D., F.A.C.P.‡

In 1890 Taylor[1] described a nodular, lobulated, intracutaneous tumor which had been present for 15 years on the left shoulder of a 43-year-old man. This had been excised 4 times but had recurred following each procedure. Other cases followed this report, but it was not until 1924 when Darier and Ferrand[2] presented for the first time a new pathologic entity, *dermatofibromes progressifs et recidivants.* They described this lesion as a neoplastic process in the skin with a progressive course and tendency to recur after incomplete removal. One year later Hoffman[3] presented similar cases under the name of dermatofibrosarcoma protuberans which has prevailed up to the present.

Bezecny[4] in 1933 reported what was to be considered the first case of dermatofibrosarcoma protuberans with metastases to the lungs and lymph nodes proved at autopsy examination. Some authors considered this lesion to be benign while others classified it as a malignant tumor. There are relatively few case reports in the literature in which this tumor had metastasized to distant organs. The following case is presented as an example of dermatofibrosarcoma protuberans with metastases.

Report of Case

A 67-year-old man was admitted to the District of Columbia General Hospital on July 17, 1962 because of extreme inanition of unknown duration. It appeared that he had been operated upon in Virginia 4 years before for a "tumor" in the right flank. There was also a history of rather marked weight loss, loss of appetite, and a general downhill course. On physical examination an old scar and an area of erythema in the right flank were noted. A mass was palpated in the abdomen and measured 23 cm. in diameter. It was hard, fixed, nontender, and nodular, and filled the upper left quadrant extending toward the left flank and back. There was a second mass measuring 13 cm. over the lower portion of the left hemithorax posteriorly which was raised and had a hard, irregular, nodular border. Another tumor measuring 2 to 3 cm. in diameter was found in the upper one-third of the right thigh. It was movable, multinodular, hard, nontender, and located subcutaneously. There was no adenopathy. The rest of the physical examination was within normal limits. Laboratory data revealed a hematocrit of 39 per cent and a white blood cell count of 8,250 with a normal differential. The platelet count was 300,000 per cu. mm., the sedimentation rate was 45 mm. per hour corrected, and the reticulocyte count was 0.6 per cent. A urinalysis was normal. Electrolytes were within normal limits except for the sodium which was found to be 129 mEq. The blood urea nitrogen was 23 mg. per cent and the fasting blood sugar 69 mg. per cent. A VDRL slide was negative. The blood uric acid was 2.5 mg. per cent, calcium 9 mg. per cent, and phosphorus 3.2 mg. per cent. All liver function tests were within normal limits including the alkaline phosphatase. The chest X-ray film revealed 2 rounded densities in the lower portion of the right lung field. A flat plate of the abdomen showed a soft tissue density in the left upper quadrant. The electrocardiogram revealed sinus arrhythmia with frequent premature ventricular contractions.

It was the impression of the house staff on admission that this patient had a hypernephroma with metastases to the lung. An excisional surgical biopsy of the mass on the posterior hemithorax was performed and a biopsy was also taken from the left thigh lesion. The pathologist reported these lesions as showing poorly circumscribed and nonencapsulated tumor composed of interweaving bundles of cells having nuclei which were oval to fusiform. They were not hyperchromatic, and there was a scanty amount of cytoplasm. Reticulum and collagen were infiltrating the adjacent fat and subcutaneous tissue. The final diagnosis was recurrent dermatofibrosarcoma protuberans.

During the patient's hospitalization a swelling of the left side of the jaw and face developed, and a 4 cm. nodule became palpable below the left mandible. On August 9, 1962 the patient was found dead in bed.

Autopsy findings: The body was that of a cachetic white man weighing about 100 pounds. A firm nodular mass measuring 2 to 3 cm. in diameter was noted on the

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*This study was supported in part by USPHS Grant CA-02824.

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inferior surface of the midportion of the left mandible. There was a hard mass measuring about 4 cm. adjacent to the left lobe of the thyroid. In the left upper quadrant of the abdomen a firm mass measuring approximately 12 by 9 cm. was noted. In the left thigh there was a 6 by 4 cm. mass which was firm and movable. The skin overlying this mass was erythematous. The left upper quadrant mass was causing an extrinsic compression of the descending colon and surrounded the left kidney, which could be separated from the lesion. There were several irregular hard nodules measuring up to 3 cm. in diameter in both lungs which on cut section were greyish-white. The left lobe of the thyroid gland was partially attached to a firm, white, glistening mass which was described previously.

The liver weighed 1,500 Gm., and on cut section showed several dark tumor-like masses. The left testicle was enlarged, measuring 6 cm. in length and 4 cm. in the AP diameter. Portions of the testicle were replaced by hard, whitish-gray tumor masses. The remainder of the autopsy was within normal limits except for the heart which revealed a partial occlusion of the left anterior descending coronary artery approximately 1 cm. from its origin.

The microscopic sections revealed the tumor masses to be very cellular and composed of broad interweaving bands of fine collagen fiber and elongated fibroblasts. The latter were fairly uniformly vesicular and had pointed uniform nuclei with apparent but not prominent nucleoli. Mitoses were few but tended to be grouped and symmetrical near the capsule. There was considerable secondary hyalinization in the deeper areas of this lesion. Fine, thin-walled vascular channels were present throughout and formed an axis for the interweaving fibers. In the lesion removed from the area of the left mandible where a section of skin was available for examination the pathologist described a highly cellular lesion separated from the adjacent compressed corium by a thin acellular hyaline-compression capsule. The nuclei of the cells were elongated and vesicular, and there were many mitotic figures. There were interlacing streams of the elongated cells. Some of the bundles were cut longitudinally and others transversely. The pattern of this lesion was identical in appearance with the histologic patterns of the biopsy specimens removed prior to death.

The final autopsy diagnosis was dermatofibrosarcoma protuberans involving the retroperitoneal area, neck, left thigh, liver, lung, left testicle, and left parotid gland. Other pathologic diagnoses were (1) arteriosclerosis with occlusion of the left anterior descending artery, (2) malnutrition secondary to malignancy, and (3) atelectasis of the upper lobe of the left lung.

Subsequent to the autopsy examination reports were obtained from Petersburg General Hospital in Virginia where the pathologist described the lesions which had been surgically excised prior to the patient's admission to D. C. General Hospital as typical of dermatofibrosarcoma protuberans. Subsequent review of these slides and those of the autopsy confirmed this finding.

Comment

Dermatofibrosarcoma protuberans is a tumor of low incidence and great controversy. Gerlach[5] in 1957 presented 32 cases recorded at the Oncologic Institute in Warsaw, Poland. He stated that until 1957 only 228 cases had been reported in the world literature, giving an incidence of 0.06 per cent. Pack and Tabah[6] collected 39 cases after reviewing some 50,000 patients seen at Memorial Hospital during the period from 1928 to 1949. Stout[7] collected 29 cases from 1907 to 1946. The variety of names applied to this lesion, the variable method of presentation, the tendency to recur, the rate of growth (up to 50 years when starting in childhood), the diversity of pathologic description, all tend to confuse the clinical picture associated with this tumor.

Stout,[7] although not personally encountering a single case of metastases in his group of 29 patients, was certain that the tumor was malignant and of the fibrosarcoma group, and he considered the grade of malignancy to be low. Pack and Tabah[6] thought the name was a misnomer and in their series did not find distant metastases. They believed some of the reported metastases could represent nonpigmented malignant melanoma. Taylor and Helwig[8] in their review of the Armed Forces Institute of Pathology file studied 115 cases and found no evidence of metastases in this group. McGregor[9] reviewed the literature briefly and reported a case of a 33-year-old colored nurse with a dermatofibrosarcoma arising in the left breast. There were movable nodules in the left axilla and the left supraclavicular fossa. Schiff in 1956[10] and in 1960[11] reported 4 cases without metastases and mentioned that there were a few reports of this lesion occurring with distant metastases but as far as he could ascertain without autopsy proof.

Metastases seem to occur late as documented by the cases reported by Binkley[12] and Gentele.[13] In the former, metastases were the cause of death 38 years after the tumor had first appeared. In the cases reported by Gentele metastases were present in the lymph nodes and in the lungs 10 years after the tumor was first discovered. Also, the 2 patients of Adams and Saltzstein[14] had a period of 14 to 20 years from the development of the tumor until their death. In his first case autopsy revealed metastases to the lung, heart, liver, lymph nodes, soft tissue, veins, and rib. The cases reported by Przybora,[15] Wooldridge,[16] and Gerlach[5] are not documented by autopsy findings, although they are clinically acceptable. Fisher and Hellstrom[17] described metastases simulating Hodgkin's disease and reticulum-cell sarcoma. They described these areas as examples of the extreme atavism which one might encounter in metastasizing dermatofibrosarcoma protuberans. Autopsy was not performed in the first case, and the other patient was alive at the time of the report. Bezecny's case[4] probably represents a metatasizing lesion. At autopsy the hilar nodes and lungs as well as the liver were involved. Photograph no. 1 of his report reveals the invasiveness of the tumor. (Taylor and Helwig[8] pose the possibility of Bezecny's tumor being a spindle-cell carcinoma.)

Penner[18] reported a case of dermatofibrosarcoma in a 3-year-old child followed from 1919 to 1947. When he died autopsy revealed pelvic, lung, pericardial, and myocardial metastases.

This partial and brief review suggests that dermatofibrosarcoma protuberans is a tumor of the fibrosarcomatous group arising in the dermis. Histologically it presents 2 microscopic types, cart-wheel and spindle-cell patterns. It has a tendency to recur with local invasiveness, and on rare occasions metastasizes via the lymphatic or blood vessels. The most common sites for metastatic lesions are the lymph nodes and the lungs. The diagnosis is a clinical one with microscopic confirmation by the pathologist.

Summary

A case of dermatofibrosarcoma protuberans with multiple metastases proved by autopsy has been reported. The original lesion appeared approximately 4 years prior to the patient's admission to the District of Columbia General Hospital. The diagnosis was based upon the clinical manifestations and confirmed by the histologic findings of a sarcoma of the spindle-cell and cart-wheel variety involving the dermis.

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Emergency Treatment of Acute Pulmonary Edema*†

PHILIP SAMET, M.D.‡

ACUTE PULMONARY EDEMA is a common, dramatic and often catastrophic event in the life history of the cardiac patient. It is primarily an expression of disease of the left side of the heart and is therefore seen in patients with acute myocardial infarction, mitral valve disease especially mitral stenosis, aortic stenosis, aortic regurgitation, severe hypertension, and occasionally in individuals with rapid tachycardias or pulmonary embolism. Rarer types include high-altitude pulmonary edema, pulmonary edema following drowning or exposure to noxious gases, and that associated with acute renal insufficiency with overhydration. On rare occasions pulmonary edema has been associated with neurological disease including trauma and intracerebral hemorrhage.

In the previously untreated patient the cornerstones of treatment of acute pulmonary edema are sedation, usually with morphine; improvement of myocardial function by digitalization; onset of rapid diuresis with the newer diuretic agents; decreasing of venous return and right ventricular output; removal of precipitating and aggravating factors such as tachyarrhythmias, paroxysmal hypertension, arterial hypoxemia, metabolic or respiratory acidosis, and increased work of breathing; and proper positioning of the patient. These therapeutic measures are most often used concurrently rather than consecutively, especially if the pulmonary edema is severe. Formerly discussion often centered about the concept as to whether morphine or digitalis is the most important form of therapy in acute pulmonary edema. The development of the newer diuretic agents such as ethacrynic acid and furosemide has added another primary form of treatment for this condition. Generally all 3 agents are administered early in the course of therapy for severe pulmonary edema, in addition to placing the patient in the upright position unless shock is present.

Morphine is best injected intravenously over a 2- to 3-minute interval in a dose of 8–10 mg. If necessary, an additional 5–10 mg. may be given one-half hour later. In mild cases the drug may be given subcutaneously or intramuscularly. Morphine therapy alone will often terminate the mild case of pulmonary edema. If respiratory depression results from morphine administration, nalorphine hydrochloride (Nalline®) should be given intravenously in 5 mg. doses which may be repeated once or twice at 20–30 minute intervals. Morphine should be avoided in patients with a history of allergic asthma or chronic pulmonary disease, especially if the arterial carbon dioxide tension (pCO_2) exceeds 45–48 mm. Hg. Pulmonary edema in patients with cerebrovascular trauma or accidents also should not be given morphine. Morphine therapy may result in varying degrees of hypotension which may in turn require therapy.

Improvement of myocardial contractility by means of digitalis is a second foundation of therapy. Intravenous therapy is generally advisable. If the situation is extremely grave and if the physician is experienced in the use of ouabain, 0.5 mg. of this glycoside is given by vein and followed by further doses of 0.1 mg. every half hour to a maximum of 0.8–1.0 mg. or until clinical improvement is noted. One must be certain that the patient has not been receiving digitalis therapy for at least 2 and preferably 3 weeks if intravenous digitalis preparations are employed. Much more frequently lanatoside C or digoxin is utilized for rapid intravenous digitalization of patients in pulmonary edema. The time of onset of action of these agents is less than 1 hour when given intravenously. If Lanoxin® (digoxin) is used, 1.0 mg. is given intravenously, followed by 1 or 2 0.25 mg. doses if needed. Maintenance therapy with oral or intramuscular digoxin may then be employed.

The development of the rapidly acting diuretic agents, ethacrynic acid and furosemide, has altered the therapy of acute pulmonary edema. The use of 1–2 ml. of a mercurial diuretic intravenously was formerly advocated, but since the onset of diuresis was delayed for 4–6 hours the issue was usually decided before an effective diuresis ensued. However, administration of either ethacrynic acid or furosemide results in much more rapid diuresis. Both agents when given intravenously produce the onset of diuresis within 15–20 minutes. A liter of urine will often be produced within 60–90 minutes. Oral administration produces diuresis within 60–90 minutes. Ethacrynic acid is given in doses of 50–100 mg. intravenously. Oral therapy (100 mg.) is less desirable. Furosemide has not as yet been released for intravenous administration but is available for oral use. Theophylline derivatives such as aminophylline (0.24–0.48 Gm. in 20 ml. of solution given slowly intravenously) are advocated by some clinicians, but I have seen little evidence of a beneficial effect unless severe bronchospasm complicates the picture of pulmonary edema. Similarly, surface-tension reducing agents such as ethyl alcohol have not been of significant benefit in my hands.

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†Submitted to the MEDICAL ANNALS by the Washington Heart Association.

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Measures to decrease venous return are employed, especially when the above approaches are not effective or are only partially effective. The primary measures in this category include the use of rotating tourniquets and phlebotomy. Phlebotomy is accomplished by removal of 500 ml. of blood as rapidly as possible. Positive pressure breathing may also be employed to decrease venous return. The blood pressure must be watched carefully during positive-pressure breathing to prevent serious hypotension. Other measures to decrease venous return, such as spinal anesthesia and stellate-ganglion blockade, have been suggested to decrease venous return but have seen limited use. Ganglionic blocking and sympatholytic agents such as mecamylamine (1–3 mg. diluted in 50 ml. of 5 per cent glucose in water given slowly intravenously), hexamethonium, dibenamine, and dihydroergokryptine have been employed, but experience has been limited. The one situation in which these agents may be useful is in the treatment of pulmonary edema in patients with severe systemic hypertension.

The last category of therapeutic measures embodies the removal of precipitating factors. These include the correction of arrhythmias with rapid or slow ventricular rates. Arterial blood gas-analysis has become an essential aspect of therapy in patients in pulmonary edema who do not readily respond to treatment. The most common acid-base abnormality detected is respiratory alkalosis which does not require specific therapy. Decreased peripheral perfusion due to low cardiac output is often a cause of metabolic acidosis, which requires correction with intravenous sodium bicarbonate. Occasionally endotracheal intubation or tracheostomy is required for adequate carbon dioxide exchange. Correction of arterial hypoxemia is accomplished by inhalation of high oxygen mixtures. The role of hyperbaric chambers for the correction of arterial oxygen unsaturation remains for future study. Surgical correction of aortic and mitral valve disease may be needed to prevent recurrent pulmonary edema. The final precipitating factors of pulmonary edema that may readily be dealt with involve restriction of activity and prevention of excessive sodium intake.

Utilization of one of the varied types of assisted circulation such as veno-arterial shunting, left-heart bypass, or counterpulsation offers future promise for the therapy of patients with nonresponsive pulmonary edema, but the clinical value and role of these methods have not yet been determined. On the other hand, peritoneal dialysis with hypertonic glucose solution offers a more immediately available palliative procedure for a few selected patients with nonresponsive continual pulmonary edema.

President's Page

DRUG ADDICTION—A DANGEROUS MENACE

An increasing menace to the health of our highly geared society is a tendency to depend more and more upon tension-releasing or stimulating drugs. Rarely can a physician in active practice go through a day in his office without prescribing drugs for sleep, tranquility, relief from depression, or detachment from the problems of life. A number of patients will ask again and again for refills of prescriptions for barbiturates, tranquilizers, and amphetamines. It is this group of repeaters that may be regarded as drug-dependent. The combined effects of alcohol and tranquilizers and the suicide danger of prescribing barbiturates to unstable individuals are well known, but the effects of long-continued barbiturates and tranquilizers on the cerebral cortex are not as familiar to most physicians.

Why do young people experiment with marijuana (pot), LSD (acid), and amphetamines (speed)? Because they are bored, said one teenager, and want to try something new. Because they want the sensual thrill of a "trip," said another; to escape from reality, said a third. For whatever reason, many students in schools and colleges in the city of Washington are experimenting with these drugs. Although the use of LSD seems to be declining, the danger to chromosome structure following the use of this powerful mood-changer is becoming apparent.

Approximately 1,100 hard-core narcotic addicts are known to live in the Washington Metropolitan Area. Probably the actual number is nearer 2,000. Heroin (smash) is the most common drug, but cocaine (snow) and dilaudid are also available. There is no law against the use of drugs, but individuals may be arrested for buying, trading, selling or giving away narcotics, including marijuana and LSD. It is a violation of the law to possess narcotics except LSD. A person may possess LSD for himself or a member of his family, but it is illegal for him to sell it. There is no law requesting a citizen to report the use of narcotics by another individual.

The Committee on Drug Dependency of the Medical Society has been engaged in evaluating the depth of drug dependency in the District of Columbia and is about to circulate to all physicians a questionnaire for this purpose. The D. C. Health Department with the United Planning Organization, using Office of Economic Opportunity funds, opened a clinic on 13th Street on March 4, 1968 for the treatment of opium-dependent persons. Here a clinical team, consisting of psychiatrists, psychologists and reformed addicts, has received a total of 176 walk-in patients. Of these, 54 were turned away because of lack of motivation, 35 have been referred to hospitals, and 87 are in the process of being evaluated.

It behooves us as members of the Medical Society to support these efforts to bring this dangerous menace under control.

William S. McCune

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by weekdays...not "cycle days"

Whether it be "shopping day," "bridge day" or "housecleaning day," a woman is accustomed to thinking in terms of days of the week rather than in "cycle days." Ovulen-21 lets her remember her natural way. Once established, her starting day is always the same day of the week... because it is fixed at three weeks on — one week off and is independent of withdrawal flow.

The same Ovulen in the same low dosage... with the same low incidence of side effects and the same high degree of protection against pregnancy.

Note: Ovulen remains available in the familiar round Compack for those women who may wish to continue to use the traditional 20-day schedule. Be sure to specify Ovulen-21 to assure each new patient of the advantages of the new *Three Weeks On—One Week Off* schedule. She might appreciate your budget-minded authorization for a six-month supply (five Refills).

Indication — For oral contraception.

Contraindications — Thrombophlebitis or a history of thrombophlebitis or pulmonary embolism, liver dysfunction or disease, known or suspected carcinoma of the breasts or genital organs and undiagnosed vaginal bleeding.

Warnings — Discontinue medication pending examination if sudden partial or complete loss of vision, or sudden onset of proptosis, diplopia or migraine occurs. Discontinue if papilledema or retinal vascular lesions occur. Since the safety of Ovulen in pregnancy has not been established, rule out pregnancy before a patient who has missed two consecutive menstrual periods continues the tablets. Consider the possibility of pregnancy at the first missed withdrawal flow if the recommended schedule has not been followed. The active ingredients in oral contraceptives have been identified in the milk of mothers receiving these drugs. The significance of this to the infant has not been determined.

Precautions — The pretreatment physical examination should specifically include the breasts, pelvic organs and a Papanicolaou smear. Endocrine and possibly liver function tests may be affected by Ovulen. Such tests should be repeated two months after stopping the medication if their results were abnormal in a woman taking Ovulen. Pre-existing fibroids may enlarge under the influence of progestin-estrogen preparations. Patients with epilepsy, migraine, asthma, cardiac or renal dysfunction, which conditions might be influenced, require careful observation because Ovulen may cause some degree of fluid retention.

Ovulen should be used with caution in patients with a history of cerebrovascular accident. Nonfunctional causes should be considered if breakthrough bleeding occurs. Adequate diagnostic measures are indicated in women with undiagnosed vaginal bleeding. Carefully observe patients with a history of psychic depression and discontinue the medication if depression recurs to a serious degree. Any possible influence of prolonged Ovulen use on pituitary, ovarian, adrenal, hepatic or uterine function awaits further study. Carefully observe diabetic patients during Ovulen use since a decrease in glucose tolerance has occurred in a few such patients. Physicians should be alert to the earliest manifestations of thrombophlebitis and pulmonary embolism since such conditions occasionally occur in patients taking oral contraceptives. Use Ovulen judiciously in young patients in whom bone growth is not complete because of the effects of estrogens on epiphyseal closure. Age is no absolute limiting factor, although Ovulen use may mask the onset of the climacteric. Pathologists should be informed of Ovulen use when relevant specimens are submitted.

Side effects — The following adverse reactions have been observed in varying incidence in patients taking oral contraceptives: nausea, vomiting, gastrointestinal symptoms (such as abdominal cramps and bloating), breakthrough bleeding, spotting, change in menstrual flow, amenorrhea, edema, chloasma or melasma, breast changes (tenderness, enlargement, secretion), change in weight (increase or decrease), changes in cervical erosions and secretions, suppression of lactation when used immediately post partum, cholestatic jaundice, migraine, allergic rash, rise in blood pressure in susceptible individuals and mental depression.

Although the following side effects have been reported in users of oral contraceptives no cause and effect relationship has been established: anovulation post treatment, premenstrual-like syndrome, changes in libido, changes in appetite, cystitis-like syndrome, headache, nervousness, dizziness, fatigue, backache, hirsutism, loss of scalp hair, erythema multiforme and nodosum, hemorrhagic eruption and itching. Thrombophlebitis, pulmonary embolism and neuro-ocular lesions have occurred in users of oral contraceptives, although a cause and effect relationship has been neither established nor disproved.

The following laboratory results may be altered by oral contraceptives: Bromsulphalein® and other hepatic function tests—increased; coagulation tests, including prothrombin, Factors VII, VIII, IX and X — increased; thyroid function — increase in protein-bound iodine and butanol extractable protein-bound iodine, and a decrease in T^3 values; metyrapone test and pregnanediol determinations.

Dosage and administration — One tablet of Ovulen-21 daily for 21 consecutive days, beginning five days after the onset of a menstrual flow (the first day of menstruation is counted as day 1), then discontinued for one week. If Ovulen is started later than day 5 after menses begins another method of protection is used until the first seven tablets have been taken. Subsequent 21-day courses are begun on the eighth day after the last tablet was taken in the preceding cycle. This three weeks on — one week off schedule is continued whether or not withdrawal flow has begun, flow has ceased or spotting or breakthrough bleeding has been experienced.

If one tablet is missed it is to be taken as soon as it is remembered and the next tablet at the usual time. If two consecutive tablets are missed the dosage is doubled for the next two days, then the regular schedule is resumed. If three consecutive tablets are missed a new tablet cycle is started on the eighth day after the last tablet was taken. For the best protection in the latter two instances instruct the patient to use another method of contraception until the next seven consecutive tablets have been taken. The possibility of ovulation increases with each successive tablet missed.

Postpartum administration — Non-nursing mothers may begin Ovulen immediately after delivery and nursing mothers after lactation is well established.

SEARLE *Before prescribing see Detailed Product Information.*
G.D. Searle & Co., P.O. Box 5110, Chicago, Illinois 60680

Ovulen-21®

Each tablet contains ethynodiol diacetate 1 mg., mestranol 0.1 mg.

three weeks on...one week off

In the complex picture of moderate to severe anxiety...

there is a new reason for prescribing Mellaril (Thioridazine HCl)

effectiveness in mixed anxiety-depression

Long recognized for its usefulness in the treatment of moderate to severe anxiety, Mellaril is now also known to be effective against mixed anxiety-depression.

Often the symptoms of anxiety states are difficult to sort out—even with the most careful probing. The patient may manifest symptoms of agitation, restlessness, insomnia, somatic complaints. But what of the depression that may be mixed in the total picture? It is reassuring to know that Mellaril may be prescribed—with strong possibilities of success—when there is anxiety alone or a mixture of anxiety and depression.

Before prescribing or administering, see Sandoz literature for full product information, including adverse reactions reported with phenothiazines. The following is a brief precautionary statement.
Contraindications: Severe central nervous system depression, comatose states from any cause, hypertensive or hypotensive heart disease of extreme degree.
Warnings: Administer cautiously to patients who have previously exhibited a hypersensitivity reaction (e.g., blood dyscrasias, jaundice) to phenothiazines. Phenothiazines are capable of potentiating central nervous system depressants (e.g., anesthetics, opiates, alcohol, etc.) as well as atropine and phosphorus insecticides. During pregnancy, administer only when necessary.
Precautions: There have been infrequent reports of leukopenia and/or agranulocytosis and convulsive seizures. In epileptic patients, anticonvulsant medication should also be maintained. Pigmentary retinopathy may be avoided by remaining within the recommended limits of dosage. Administer cautiously to patients participating in activities requiring complete mental alertness (e.g., driving). Orthostatic hypotension is more common in females than in males. Do not use epinephrine in treating drug-induced hypotension. Daily doses in excess of 300 mg. should be used only in severe neuropsychiatric conditions.
Adverse Reactions: *Central Nervous System*—Drowsiness, especially with large doses, early in treatment; infrequently, pseudoparkinsonism and other extrapyramidal symptoms; nocturnal confusion, hyperactivity, lethargy, psychotic reactions, restlessness, and headache. *Autonomic Nervous System*—Dryness of mouth, blurred vision, constipation, nausea, vomiting, diarrhea, nasal stuffiness, and pallor. *Endocrine System*—Galactorrhea, breast engorgement, amenorrhea, inhibition of ejaculation, and peripheral edema. *Skin*—Dermatitis and skin eruptions of the urticarial type, photosensitivity. *Cardiovascular System*—Changes in the terminal portion of the electrocardiogram have been observed in some patients receiving the phenothiazine tranquilizers, including Mellaril (thioridazine hydrochloride). While there is no evidence at present that these changes are in any way precursors of any significant disturbance of cardiac rhythm, several sudden and unexpected deaths apparently due to cardiac arrest have occurred in patients previously showing electrocardiographic changes. The use of periodic electrocardiograms has been proposed but would appear to be of questionable value as a predictive device. *Other*—A single case described as parotid swelling.

Mellaril®
(Thioridazine HCl)
25 mg. t.i.d.

for moderate to severe anxiety and mixed anxiety-depression

SANDOZ SANDOZ PHARMACEUTICALS, HANOVER, N. J. 68-16

Editorials

Clinical Laboratory Performance in the United States

Clinical pathology laboratories are one of the cornerstones of medical practice today, with more than 500 million clinical laboratory tests being performed annually in hospital and independent laboratories. Laboratory medicine is helping physicians save hundreds of American lives daily. With the increasing dependence of physicians on laboratory determinations for diagnosis and therapy it is essential that such results be consistently reliable. To this end, clinical pathology has voluntarily established a system of self-appraisal, self-criticism, and self-discipline quality control.

Yet, since February 11, 1966 a smokescreen of untrue statements, exaggerated charges, incorrect statistics, misleading reports, and incredible claims has been circulated in the search for acceptance by the uninformed. The misleading and biased information originated at a subcommittee hearing of the House of Representatives on testimony of the Department of Health, Education, and Welfare. The testimony was written material submitted by the Public Health Service attempting to justify a funding request for the "National Laboratory Improvement Program." It stated: "Premature death, extended hospital stay, unnecessary suffering, and loss of productivity as well as tremendous economic losses are consequences of inaccurate laboratory results. It is conservatively estimated that at least 430 million tests are performed annually with an approximate error rate of 25 per cent. At an average cost of $4 per test this represents a waste of some $430 million annually to the Nation in payment for erroneous laboratory diagnostic results. The cost in human life and suffering is beyond monetary measurement."

No one could deny the intent of such a statement. The National Communicable Disease Center's representatives and HEW officials presented no documentation in support of the statement, apparently believing that it was the expression of a self-apparent truth.

As a result, an Ad Hoc Advisory Panel of the National Communicable Disease Center was formed and subsequently submitted to a House Committee the study, "A Report on Laboratory Performance and Methods of Improvement." The report was an attempt to give credence to the previous statement. To do this the Committee went to an obvious, misrepresentative, error-ridden segment of "the Nation's clinical laboratories," i.e., 5 NCDC surveys, a survey from New York City, a survey from Canada, and 2 surveys, 15 and 20 years old, respectively. The scope of the NCDC surveys covered small surveys of governmental laboratories such as state health departments, Indian Bureau hospitals, and Federal prisons. Such a report, purporting to reflect the "Nation's clinical laboratories," is apparently an accurate appraisal of governmental controlled laboratory systems.

In contrast to the report's findings, former President of the College of American Pathologists, Dr. Ernest E. Simard, points out that in the 1965 National Comprehensive Survey, Clinical Chemistry Section of the CAP Standards Committee, 92 per cent of 12,606 tests on 1,146 samples in 1,146 clinical pathology laboratories were medically useful. A CAP Small Hospital Survey in 1966 showed ABO blood grouping to be 95 per cent accurate and Rh typing to be 96 per cent accurate. Current reports reveal an incidence of hemolytic transfusion reactions from 0.01 to 0.03 per cent, or approximately 500–1,500 per year, in contrast to the report's figures of 50,000–250,000.

The report's gross misstatements are not only objectionable but are also a travesty of the truth. They should be expunged as they are invalid criteria for evaluation of laboratory performance.

The record shows that quality control procedures were begun in clinical pathology laboratories in hematology, histopathology, and serology in the 1920's and 1930's; in blood banking in the 1940's; and in clinical chemistry in 1950. Thou-

Opinions expressed in contributions to the Editorial Section are those of the writers and do not necessarily reflect the views of the Medical Society of the District of Columbia

Narcotic Don'ts for the Physician

DON'T leave prescription pads around.
—Addicts want them for effecting narcotic forgeries.

DON'T write a narcotic prescription in lead pencil.
—Avoid writing any prescriptions in pencil; many are changed to call for morphine.

DON'T prescribe narcotics with numbers such as 10 or X without the use of brackets or parentheses.
—The units of narcotics should be bracketed so that they cannot be increased by the addition of a zero or an X.

DON'T carry a large stock of pads or narcotics in your bag.
—Addicts are on the lookout for these in doctors' offices and cars.

DON'T store your office supply of narcotics where patients can see and have access to it.
—Avoid storage near sink or urinal. The patient may ask to use these and can steal drugs at that time.

sands of pathologists and medical technologists have been trained in quality control. The College of American Pathologists' standards require quality control in the Inspection and Accreditation Program. The Commission on Continuing Education of the American Society of Clinical Pathologists has conducted workshops, published manuals, and produced instructional film strips on quality control. These efforts in external and internal quality control are important and attest to the fact that quality control is extensively used in the clinical pathology laboratories of the Nation.

Obviously, laboratory performance throughout the United States is far better than the contrary opinion of the report. When medical usefulness is used as an evaluation criterion, more than 95 per cent of laboratory results in the United States are acceptable and support correct medical decisions. While some might like to say that there are no errors in laboratory tests, pathologists know that there are and probably always will be. Advances in the past 15 years have led to the recognition and measurement of the variables causing errors. Most physicians realize that "false negative" and "false positive" test results occur in 1 to 10 per cent of some tests, but they realize that these are not laboratory errors. Rather, they are limitations of interpretation and not performance.

OSCAR B. HUNTER, JR., M.D.
President, College of American Pathologists

The Consultation*

FORTY YEARS AGO I witnessed for the first time a consultation, and it has been a vivid memory ever since. My mother was ill with pneumonia, our physician was Dr. C. H. Kleinschmidt, a member of this Society. He called in consultation Dr. Louis Mackall, the father of our Dr. Louis Mackall, Jr. Those 2 physicians met in a dignified and friendly way. A few words passed between them and then the family physician led the way to the bedside. The consultant carefully examined the patient and the records. Then those 2 earnest, serious-minded men withdrew behind closed doors, and I could hear their voices really in consultation, and then they called us into the room and our own family physician, in the presence of the consultant, told us of the results of their consultation. My mother died, but there was never a moment of doubt in our minds but that she had received all of the knowledge and skill of those 2 able physicians.

That consultation was a ceremony that inspired the utmost faith. How different are many of the consultations of today, for often a consultant is asked to go and examine a patient in the absence of the physician and then to telephone his findings. A few years ago I was called in consultation. Several physicians were there. We had a long conference before I saw the patient, but before I had finished my examination they had gone. My opinion was not wanted.

I may be old-fashioned, but I believe in the ritual of the consultation. I believe in the value of details, and the details of a properly performed consultation do much to maintain the respect, regard, and confidence of the patient in the art of medicine and the physician, which is so essential to the successful practice of the healing art.

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* From the Presidential Address of Dr. A. B. Bennett, March 20, 1935. Dr. Bennett died, June 9, and his obituary appears in this issue of the ANNALS.

The Hospital for Sick Children

By official action of the Board of Managers, the name of the Children's Convalescent Hospital has been changed to The Hospital for Sick Children. The decision to change the name grew out of a desire to more accurately describe the Hospital's nature and function. Located at 1731 Bunker Hill Road, N.E., it is Washington's only hospital dedicated solely to the care of children suffering from illnesses requiring prolonged diagnosis and therapy. It was founded in 1883 as the Children's Country Home and has been known since 1956 as the Children's Convalescent Hospital.

The medical services of the Hospital cover chronic respiratory conditions including asthma, emphysema, chronic bronchitis, and cystic fibrosis, which has recently come into focus as a specific entity in the community. A special program is underway for the residential care of children at the Hospital in addition to the continuing focus for the treatment of across-the-board chronic illnesses, which include meningitis, heart, kidney, metabolic disorders, neuromuscular problems, multiple congenital anomalies, and illnesses involving the speech, sensory, and psychic systems.

In order to help heal the afflicted children, a multidisciplinary approach is employed which emphasizes psychological and social needs as well as those of medical and biological.

Fully accredited by the Joint Commission on Accreditation of Hospitals, the Hospital is a member of the American Hospital Association, the Maryland-District of Columbia-Delaware Hospital Association, the Hospital Council of the National Capital Area, and the Health and Welfare Council of the National Capital Area. Served by a large mdical staff in appropriate specialties, the Hospital provides 50 beds for pediatric care. A new building now under construction will increase the bed capacity to 85. If a child may benefit from the comprehensive services available, he may be admitted. There are no restrictions as to residence, race, or ability to pay. Referrals for admission come from community agencies, other hospitals, and private physicians. The Hospital has developed a close working relationship with all of these sources of admission, and it continually strives to maintain this rapport.

On March 28 the Hospital became affiliated with Georgetown University School of Medicine for the development of educational and training programs. The Hospital has contracts for services with the District of Columbia Health and Welfare Departments, Montgomery and Prince George's Counties in Maryland, Maryland Crippled Children's Bureau, Fairfax County, and the City of Alexandria. It also has a contract with the Comprehensive Health Care Program located at Children's Hospital. The Hospital attempts to collect for its charges either through a third party carrier, if applicable, or from the family, depending upon ability to pay.

In the new building, which has just been completed, extensive hospital facilities will be provided, including recreation facilities, in keeping with our unique philosophy of providing a homelike atmosphere in a highly specialized, fully equipped hospital for sick children.

Frederic G. Burke, M.D.
Medical Director
Warren R. Betts
Administrator

► HEALTH DEPARTMENT NOTES

Nuclear Medicine Division at D. C. General Hospital

Although the Nuclear Medicine Division at D. C. General Hospital opened officially only last month (July), limited services have been available there since January. Numerous tests utilizing radioactive isotopes have been made, most frequently involving organ scans and thyroid function studies.

Diagnostic procedures involving nuclear medicine at D. C. General Hospital include volume or quantity determinations, function tests, and organ scans. Maximum diagnostic information from these procedures is available when the clinician responsible for the procedure is fully aware of the patient's total medical profile.

Nuclear medicine may be defined as the discipline concerned with the diagnosis, treatment, and clinical investigation of disease by utilization of internally administered radionuclides and radionuclide-labeled medicinals. Diagnoses and treatment with radioisotopes are considered safe, rapid and accurate. The remarkable sensitivity of the instrumentation permits the use of trace amounts of radioactive material for diagnostic purposes. The amount of exposure, in most cases, is less than that received in a routine chest X-ray.

Clinical radioisotope procedures are provided in the Nuclear Medicine Division both for D. C. General Hospital patients and individuals referred there from Health Department clinics. Division staff members stress the importance of an informative session with the patient and possibly the family before starting a procedure involving the use of radioactive material. An explanation of the procedure and the benefits to be achieved with only minimal exposure to radiation should be included and pertinent questions answered.

While the primary function of the Nuclear Medicine Division is to provide clinical services and techniques for the Hospital and clinic-referred patients, the Division has additional objectives.

The Division's educational program has both immediate and long-range elements. For the present, medical students, house officers, and staff physicians from the various treatment divisions within the Hospital will be instructed in the science of nuclear medicine. Necessary facilities and equipment for such instruction will be maintained by the Division.

The Division also is responsible for providing acceptable training and experience in nuclear medicine to those physicians seeking to obtain licensure from the Atomic Energy Commission for the use of radioisotopes within their individual practices.

The long-range phase of the educational program is concerned with the training of specialists in nuclear medicine. A definition of the educational requirements to enter a residency in nuclear medicine is being developed by the American Medical Association and representatives of the Society of Nuclear Medicine. When the definition has been established and a specialty board in nuclear medicine has been created, D. C. General Hospital's Nuclear Medicine Division will be prepared to offer residencies in this discipline.

Meanwhile a program of training grants established by the U. S. Public Health Service provides a limited number of applicants with opportunities to train for either 1 year or a 3-year period in nuclear medicine. Facilities for training under such grants are now available at D. C. General Hospital.

A third important objective of the Nuclear Medicine Division is an ongoing schedule of clinical research in techniques and therapeutics for patient care. Basic research in the new radiopharmaceutical compounds and procedures will be carried on, utilizing grants and other available funds.

Consultations with staff members from the Hospital's various treatment divisions constitute a basic and important part in the development of a clinical research program. This provides a means of improving techniques and treatment of patients. Consultant and service resources in nuclear medicine are available to personnel engaged in other areas of research at D. C. General Hospital.

The Nuclear Medicine Division also offers consultative services for all research utilizing radioisotopes in other facilities in the District of Columbia and is prepared to provide technical assistance and advice on instrumentation, experiment design, personnel safety, and record-keeping.

In all phases of its operation, the Nuclear Medicine Division is concerned with safety factors. Regulations and precautions designed to minimize radiological exposure of both patients and personnel, in accordance with AEC recommendations, are administered by the Division.

Murray Grant, M.D., D.P.H.
D. C. Director of Public Health

▶NEW MEMBERS

Active

Guillermo Alfredo Balfour, M.D. Office: 2125 13th Street, N.W. Born in Argentina. Medical degree: Buenos Aires University, 1959. Specialty: Pediatrics.

Joseph Clement Boschulte, M.D. Office: 734 Longfellow Street, N.W. Born in Tortola, British W. I. Medical degree: Howard University, 1958. Specialty: Psychiatry.

Ashraf Zakaria El-Khodary, M.D. Office: Georgetown University Hospital. Born in Egypt. Medical degree: Kasr El-Aini, Cairo, 1953. Specialty: Internal Medicine.

Francis Beneditto Fernandes, M.D. Office: 730 Maryland Avenue, N.E. Born in Port-of-Spain, Trinidad. Medical degree: Howard University, 1964. Specialty: General Practice.

Jerome Norman Goldman, M.D. Office: 1746 K Street, N.W. Born in New York City. Medical degree: New York University, 1956. Specialty: Ophthalmology.

Thomas William Moore, Jr., M.D. Office: 1520 9th Street, N.W. Born in Boston, Mass. Medical degree: University of Geneva, Switzerland. Specialty: General Practice.

Sumner Gerald Sandler, M.D. Office: Department of Hematology, Georgetown University Hospital.

Born in Washington, D. C. Medical degree: New York University, 1962. Specialty: Internal Medicine.

Nelson Perez Trujillo, M.D. Office: Walter Reed General Hospital. Born in Cuba. Medical degree: Tulane Medical School, 1962. Specialty: Internal Medicine.

Associate

Peter V. Siegel, M.D. Office: 800 Independence Avenue, S.W. Born in Syracuse, Mo. Medical degree: New York University, 1943. Specialty: Aviation Medicine.

Resident-Intern

Robert H. Birschbach, M.D. George Washington University Hospital. Born in Mount Calvary, Wis. Medical degree: Marquette University, 1963. Specialty: Internal Medicine.

Peter L. H. Tynberg, M.D. U.S. Public Health Service. Born in Pittsburgh, Pa. Medical degree: University of Pittsburgh, 1966. Specialty: General Practice.

Information Office on Clinical Therapeutic Trials on Cancer

A Committee on Clinical Therapeutic Trials was established within the scope of the Commission on Clinical Oncology at the last meeting of the International Union Against Cancer, which was held in Tokyo. The Committee has set itself 4 tasks:

1. To facilitate the exchange of information about trials.
2. To assist the coördination of coöperative trials carried out in 2 or more countries.
3. To advise on the design of trials.
4. To promote the training of investigators.

Up to now there has been no available information both on trials in progress and on completed trials where the results are unpublished. In view of the length, complexity, and cost of controlled therapeutical trials, it would help if, before starting a new trial, an investigator could obtain information not only on published literature but on trials in progress on the same or on related subjects as well.

In order to meet its responsibilities in the field of information, the Committee has established an office for the collection, registration and distribution of information. The more information transmitted to the office, the better will be the service it can provide. Such information may be sent to the office or (in the U.S.) to Dr. George A. Higgins, Jr., Chief of Surgical Service, Veterans Administration Hospital, Washington, D. C. 20422. Dr. Higgins is a member of the Committee. The address of the central office is:

Unité de Recherches Statistiques de l'I.N.S.E.R.M.
Institut Gustave Roussy
16 bis, Avenue Paul-Vaillant-Couturier
94-Villejuif, France.

Dr. Holden Elected to AMA Board of Trustees

Dr. Raymond T. Holden, the Medical Society's senior delegate to the American Medical Association, was elected to the Board of Trustees of the AMA during its 117th Annual Convention in San Francisco, June 16 to 20. Dr. Holden's qualifications for the office were presented to the House of Delegates, June 20, by Dr. John J. Hughes, junior delegate to the AMA. His nominating speech follows:

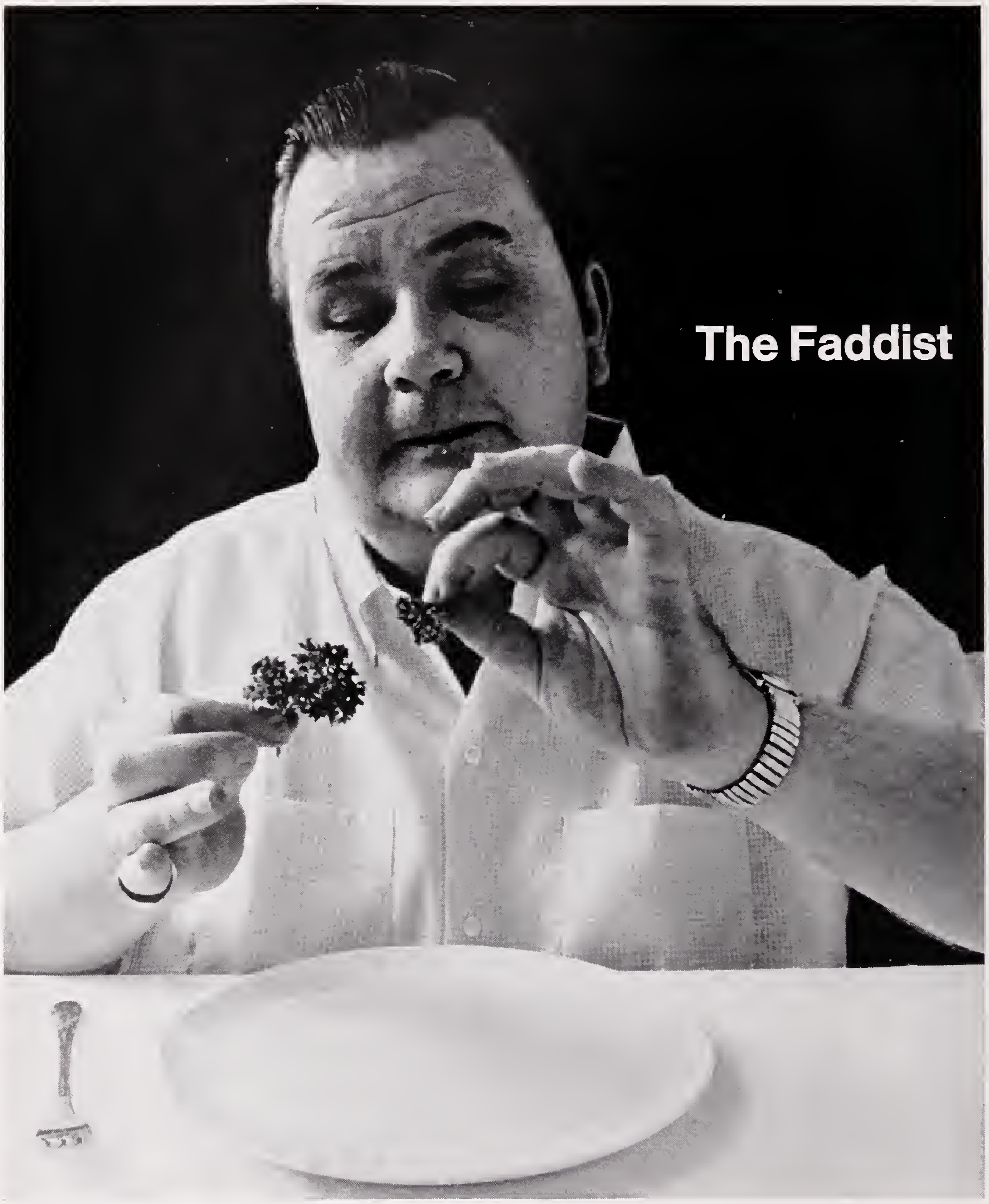
"For a position on the Board of Trustees of the American Medical Association, the Medical Society of the District of Columbia is pleased to offer a candidate who has a distinguished record of service to his Society and to the AMA.

"Our candidate, who has been in private practice in Washington since 1932, is Clinical Professor of Obstetrics and Gynecology at Georgetown University School of Medicine and has served as Chief of Staff and trustee of the Columbia Hospital for Women.

"After serving with distinction in the United States Navy during World War II he remained in the Naval Reserve and rose to the rank of Rear Admiral in 1960. He retired from the Reserve in 1966. For years he has been consultant to the Surgeon General of the Navy and to the U. S. Naval Hospital in Bethesda, Md.

"Our candidate was President of the Medical Society of the District of Columbia in 1947–48 and has served on its Executive Board since 1939. He has been our delegate to this House since 1953. In the latter capacity he has served on many reference committees and as chairman of several. His effectiveness as Chairman of the Committee on Human Reproduction is well known to this body.

"The Medical Society of the District of Columbia believes that its candidate by background and experience is exceptionally well qualified to serve on the Board of Trustees of the American Medical Association.



He's the one who plays the weighting game.

Famine, then feast. Crash diets. Pot luck.

He's the one who needs your professional help.

You can help the faddist realize there's really no fast way. And, that a healthy loss requires changing ways as well as weight. Explain the benefits of a planned program of diet, exercise, proper rest and will power. Work out a sound program to keep him physically fit.

Project Weight Watch can help, too. We have available free materials and suggested diets. Scientific diets based on the 4 food groups—meat, breads and cereals, fruits and vegetables and dairy foods. These are diets you'd write yourself if you had time. Take a minute and send for them.

PROJECT
WEIGHT
WATCH
FACTS, NOT FADS

Get him off his fad today.

Name

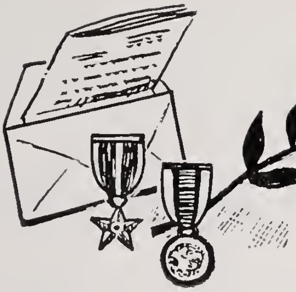
Position

Address

City

State Zip

DAIRY COUNCIL OF GREATER METROPOLITAN
WASHINGTON, D.C.
1511 K Street, N. W., Washington, D.C. 20005



News & Personals

Summary of Executive Board Actions

Proceedings of June 24 Meeting

Dinner for the Board members was served in the Exhibit Hall of the Medical Society. Dr. Edward W. Nicklas, Chairman, presided at the meeting which was attended by Drs. William S. McCune, Frank S. Bacon, Jed W. Pearson, Jr., Gudrun Augustin-Rueckert, Paul R. Wilner, Robert J. Coffey, Henry D. Ecker, John J. Hughes, and Mr. Thomas Sadler, Secretary. The meeting was called to order by Dr. Nicklas at 7:15 p.m.

The minutes of the May 27, 1968 Board meeting were approved as submitted.

Committee Meetings: The Secretary reported the following committees had met since the last meeting of the Board:

May 28 Committee on Liaison with Hospitals
29 Recruitment Subcommittee, Regional Medical Program
June 3 Committee on Medical Practice
4 Committee on Society Insurance
5 Fee Review Committee
6 Committee on the Study of Cancer
10 Committee on Mental Health
11 Board of Credentials
11 Committee on Liaison with the Clergy
11 Committee on Medicolegal Problems
12 Membership Committee
18 Grievance Committee
20 Committee on Society Insurance

Board of Credentials: On recommendation of the Board of Credentials, 8 applicants were elected to Active membership, 1 to Associate membership, and 2 to Resident-Intern membership. Their names appear on page 436. Approval was also given to the following changes in membership status:

Transfers

Active to Life: Joseph H. Cowan
Active to Associate: Eleanor L. Long
Resident-Intern to Active: John Francis Bresette

Resignation

Active: Bushnell Smith

Dr. James A. Ryan has resumed practice in the District of Columbia and, at his request, was reinstated to Active membership.

AMA Annual Convention: Dr. Nicklas asked Dr. Hughes to read to the Board his speech before the House of Delegates nominating Dr. Raymond T. Holden to the Board of Trustees of the AMA (see page 437). Dr. Hughes also described a few highlights of the AMA Convention, which was held in San Francisco, June 16–20. A formal report from the delegates will be submitted at the July meeting.

AMA Regional Legislative Conference: Dr. Pearson, who attended the recent AMA Regional Legislative Conference, stated that the AMA is trying to achieve the enactment of sound medical legislation and the defeat of undesirable proposals by continued and improved relationships between members of Congress and the medical profession. The AMA is also hoping to activate a continuing physician task force in each Congressional District to work with and through the county medical societies in that District. He commended the AMA's Council on Legislative Activities for outstanding performance in its analyses of all health-oriented bills.

Luncheon Meeting with Dr. Grant: In their most recent luncheon meeting Dr. McCune and Dr. Murray Grant had discussed the Medicaid fee schedule. Dr. Grant advised Dr. McCune that this fee schedule had been formulated by a professional subcommittee and that he would give him the names of the members of this committee as well as other health-oriented committees.

In answer to Dr. McCune's question regarding prescription refills for welfare patients, Dr. Grant stated that such patients could go not only to D. C. General but to 5 area pharmacies designated for such refills.

At this point (9:20 p.m.) the meeting was adjourned because of local conditions which had brought about a 9 p.m. curfew.

GENERAL NEWS

Georgetown University Medical Center has available **residencies in psychiatry** to qualified physicians who wish to become psychiatrists. Training stipends of $12,000 yearly are granted for the 3 years of the residency program.

To be eligible a physician must have been in practice for a minimum of 4 years following internship (which may include time in the Armed Forces or residency training in some specialty other than psychiatry). Additional requirements are American citizenship, graduation from an approved medical school, and 1 year of approved internship.

Applicants should contact Dr. Richard A. Steinbach, Department of Psychiatry, Georgetown University Medical Center, Washington, D. C. 20007.

The National Research Council, on behalf of the James Picker Foundation, is accepting applications for **postdoctoral awards of fellowships and grants in the field of radiology and nuclear medicine** for 1969-70. Awards are made in 4 categories:

1. Advanced Fellowships in Academic Radiology where preference will be given to applicants less than 35 years of age who have at least 3 years of residency or postdoctoral training in radiology or radiological sciences.
2. Research Fellowships open to recent graduates who desire investigative experience and training in radiology or nuclear medicine.
3. Grants for Scholars to assist medical schools, hospitals, and other research institutions in supporting and developing junior staff members in radiology and nuclear medicine.
4. Research Grants designed to encourage investigations in radiology and nuclear medicine offering promise of improvement in methods of diagnosis or treatment of disease.

Applications should be submitted by October 1, 1968. Application blanks and further details may be obtained from the Committee on Radiology, National Research Council, 2101 Constitution Avenue, N.W., Washington, D. C. 20418.

Dr. Leonard T. Peterson was elected Chairman of the Board of Trustees of **Medical Service of the District of Columbia** at the June meeting of the Board. Mr. George B. McGovern was elected President. As Chairman of the Board, Dr. Peterson will be the chief executive officer while Mr. McGovern will be the chief administrative officer. Dr. Peterson formerly served as President of the Plan and Mr. McGovern as Executive Vice President.

Dr. Jack Kleh was elected Vice Chairman of the Board. Mr. Alan F. Ridgway was appointed Executive Vice President and elected Secretary of the Plan. Reëlected Treasurer was Mr. Douglas R. Smith, Chairman of the Board and President of National Savings and Trust Company.

Three new trustees elected to the Board for 3-year terms were Dr. Howard A. Donald and Dr. Russell M. Tilley, Jr., representing the Medical Society of D. C., and Dr. Leon R. Levitsky, representing Prince George's County (Md.) Medical Society. Dr. Levitsky is an Associate member of the D. C. Medical Society.

Dr. Herbert L. Ley, Jr., who has been Director of the Food and Drug Administration's Bureau of Medicine, was appointed to succeed Dr. James L. Goddard as **Commissioner of Food and Drugs.** Dr. Goddard recently resigned, effective July 1, to join a data processing firm in Atlanta, Ga. Dr. Ley, a graduate of

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A simplified approach to the practical management of hypertension

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Enduron: (methyclothiazide) A basic building block for mild hypertensives

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100 No. 6812
Tablets (Grooved)
ENDURON
METHYCLO-
THIAZIDE
5 mg

Excellent day-long Na^+ output, yet easy on the K^+

Enduron provides an excellent starting therapy. Your patient's sodium excretion is greatly enhanced. Yet potassium loss is low.

The therapeutic action is smooth, and persists for a full 24 hours. With Enduron you can prescribe convenient once-a-day dosage without skimping your patients on day-long thiazide effectiveness.

Of course, as with all thiazides, supplemental dietary potassium should also be considered.

Use Enduron as a basic therapy in patients with mild to moderate hypertension. A single 5-mg. tablet each day is ample in most cases.

Once a day, every day
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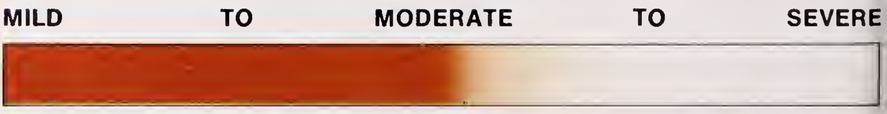
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Enduronyl: Its deserpidine component adds response in moderate hypertension

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Tablets (Grooved)
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Each tablet contains
Methyclothiazide 5 mg
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Less frequent rauwolfia side effects than with reserpine

When you wish to build further response, consider shifting to Enduronyl.

Enduronyl adds a building block of *deserpidine*. This is a purified rauwolfia alkaloid available only from Abbott. It adds good antihypertensive and tranquilizing activity. Yet its incidence of untoward effects, particularly lethargy and depression, is lower than with reserpine.

Enduronyl is available plain or Forte. The latter provides its variation where most helpful, by doubling the deserpidine.

Use Enduronyl for patients throughout the broad range of mild to moderately severe hypertension.

Once a day, every day

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METHYCLOTHIAZIDE 5 mg. with
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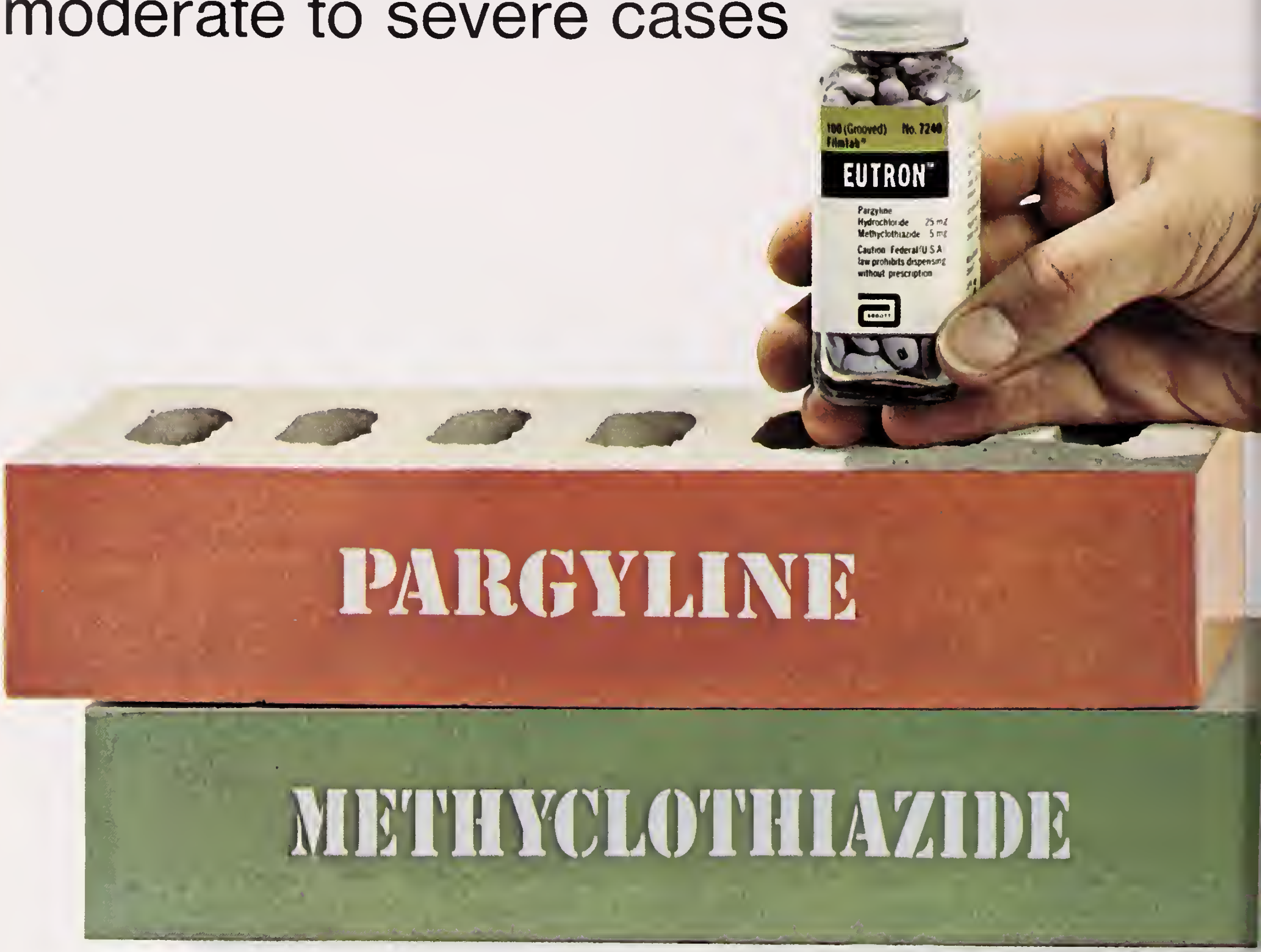
Eutron: A unique combination for handling moderate to severe cases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METHYCLOTHIAZIDE

Affords almost uniform diastolic reduction in all body positions

Eutron lowers diastolic pressures nearly equally, whether your patient is standing up or lying down.

Thus, in clinical trials, average *standing* diastolic readings were reduced from 112 pre-treatment to 90 post-treatment; *sitting* from 115 to 95; and *recumbent* from 112 to 94.

Note that following Eutron, the diastolic reductions were nearly alike in all three body positions.

Use Eutron for managing your moderate to severe cases. Its building blocks enhance each other; hence lesser doses often suffice.

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PARGYLINE HYDROCHLORIDE 25 mg.
with METHYCLOTHIAZIDE 5 mg.

MILD
TO
MODERATE
TO
SEVERE

See Brief Summary on final page of advertisement

801094

ENDURON®
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ENDURONYL®
Each tablet contains
Methyclothiazide 5 mg. with
Deserpidine 0.25 mg. or 0.5 mg.

Indications: Edema and mild to moderate hypertension (Enduron), and mild to moderately severe hypertension (Enduronyl). More potent agents, if added, can be given at reduced dosage.

Contraindications: Sensitivity to thiazides; severe renal disease (except nephrosis) or shutdown; severe hepatic disease or impending hepatic coma (hepatic coma due to hypokalemia has been reported in patients on thiazides). Do not use Enduronyl in severe mental depression, suicidal tendencies, active peptic ulcer, or ulcerative colitis.

Warnings: Consider possible sensitivity where there is history of allergy or asthma. If added potassium is indicated, dietary supplementation is recommended. Reserve enteric-coated potassium tablets for cautious use only when necessary, as they may induce serious or fatal small bowel lesions (stenosis with or without ulceration), cause obstruction, hemorrhage, and perforation often requiring surgery; discontinue them immediately if abdominal pain, distention, nausea, vomiting, or g.i. bleeding occurs. Neither Enduron nor Enduronyl contains added potassium.

Precautions: Use thiazides cautiously in severe renal dysfunction, impaired hepatic function or progressive liver disease; also in pregnancy (bone marrow depression, thrombocytopenia, and altered carbohydrate metabolism have been reported in certain newborn). In surgery, thiazides may reduce response to vasopressors, and increase response to tubocurarine. Antihypertensive response may be enhanced following sympathectomy. Watch for electrolyte imbalance (e.g., hyponatremia) in all patients. In hypokalemia (especially in digitalized patients) give supplemental potassium. In hypochloremic alkalosis, give supplemental chloride.

Use rauwolfias with caution in patients with history of peptic ulcer. Rauwolfias with anesthetics may produce hypotension and bradycardia. Discontinue Enduronyl two weeks before elective surgery. Consider vagal blocking agents during emergency surgery. In epilepsy, adjust anticonvulsant dosage. In electroshock, shorten stimulus strength and duration. In occasional patients with depressive tendencies, rauwolfias may precipitate severe mental depression that usually disappears when drug is stopped.

Adverse Reactions: Thiazide reaction include blood dyscrasias (thrombocytopenia with purpura, agranulocytosis, aplastic anemia); elevation of BUN, serum uric acid or blood sugar; anorexia, nausea, vomiting, diarrhea, headache, dizziness, paresthesia, weakness, skin rash, photosensitivity, jaundice, symtomatic gout, and pancreatitis. Cutaneous vasculitis in the elderly has been reported with other thiazides. Adverse effects with deserpidine are qualitatively similar to those with reserpine, but their incidence is lower. These include nasal stuffiness, abdominal cramps or diarrhea, nausea, headache, weight gain, reduced libido and potency, peptic ulcer aggravation, epistaxis, skin eruption, asthma in susceptible patients, electrolyte imbalance, excessive salivation, and a reversible Parkinson's syndrome. Excessive drowsiness, fatigue, weakness, and nightmares may signal mental depression. Thrombocytopenia, purpura, and a symptom manifested by dull sensorium, deafness, uveitis, glaucoma, and optic atrophy are rare allergic reactions to other rauwolfias. Hypotension from antihypertensive agents may precipitate angina attacks in susceptible individuals. Usually adverse reactions disappear when drug is withdrawn.

EUTRON™
Each tablet contains
Pargyline Hydrochloride 25 mg.
with Methyclothiazide 5 mg.

Indications—Moderate to severe hypertension.

Contraindications—Pheochromocytoma, paranoid schizophrenia, hyperthyroidism and advanced renal failure. Not recommended in malignant hypertension, children under 12, pregnant patients.

Do not use with: centrally or peripherally acting sympathomimetic drugs; foods high in tyramine (e.g., aged and natural cheeses); parenteral reserpine or guanethidine; imipramine, amitriptyline, desipramine, nortriptyline or their analogues; other monoamine oxidase inhibitors; methyldopa or dopamine; separate Eutron and these agents by two weeks.

Sensitivity to thiazides; severe renal disease (except nephrosis) or shutdown; severe hepatic disease; impending hepatic coma from thiazide-induced hypokalemia.

Warnings—Patients: 1. No other drugs (particularly "cold preparations" and antihistamines), cheese or alcohol without physician's consent. 2. Promptly report orthostatic symptoms, severe headache, other unusual symptoms. 3. Angina pectoris or coronary artery disease patients must not increase physical activity with improved anginal symptoms or well-being.

Physicians: 1. Use antihistamines, hypnotics, sedatives, tranquilizers and narcotics (meperidine contraindicated) cautiously in reduced doses. 2. Stop Eutron two or more weeks before elective surgery; in emergency surgery reduce premedication (narcotics, sedatives, analgesics, etc.) to 1/4 to 1/5; carefully adjust anesthetic dosage to patient response. 3. Use cautiously in advanced renal failure. 4. Pargyline may induce hypoglycemia. 5. Consider possible sensitivity reactions when a history of allergy or asthma is present. 6. If potassium is indicated, dietary supplement is recommended; enteric-coated potassium tablets may induce serious or fatal small bowel lesions (stenosis with or without ulceration), cause obstruction, hemorrhage, and perforation frequently requiring surgery; discontinue medication immediately if abdominal pain, distention, nausea, vomiting or gastrointestinal bleeding occurs; Eutron does not contain added potassium. 7. Possible systemic lupus erythematosus has been reported for thiazides.

Precautions—Pargyline: Use cautiously at reduced dosage: caffeine, alcohol, antihistamines, barbiturates, chloral hydrate, other hypnotics, sedatives, tranquilizers, narcotics. Periodically do urinalyses, blood counts, liver function tests, etc. Use with caution in liver disease. Watch for orthostatic hypotension, especially in impaired circulation (e.g., angina pectoris, coronary artery disease, cerebral arteriosclerosis); also, augmented hypotension in concomitant febrile illnesses. Reduce or discontinue if hypotension is severe. In impaired renal function watch for cumulative drug effects, elevated BUN and other evidence of progressive renal failure; withdraw drug if these persist. In surgery increased central depressant response (hypotension and increased sedative effect) can be controlled by (1) discontinuing at least two weeks prior; (2) in emergency surgery lowering dose of premedication; (3) when necessary, administering a vasopressor. Do not use in hyperactive and hyperexcitable patients. Pargyline may unmask severe psychotic symptoms where emotional problems pre-exist. Use cautiously in Parkinsonism, especially with antiparkinsonian agents. In prolonged therapy, examine for change in color perception, visual fields, fundi and visual acuity. Also, prolonged therapy has made certain patients refractory to nerve blocking effects of local anesthetics.

Methyclothiazide: Use cautiously in severe renal dysfunction, impaired hepatic function or progressive liver disease; also in pregnancy (bone marrow depression, thrombocytopenia, and altered carbohydrate metabolism have been reported in certain newborn). In surgery thiazide may reduce vasopressor response and increase tubocurarine response. Antihypertensive response may be enhanced following sympathectomy. Watch for electrolyte imbalance (e.g., hyponatremia). Give supplemental chloride if hypochloremic alkalosis occurs and supplemental potassium if hypokalemia occurs (especially in digitalized patients). Thiazides may decrease serum P.B.I. without signs of thyroid disturbance.

Adverse Reactions — Pargyline: Orthostatic hypotension and associated symptoms, mild constipation, fluid retention, edema, dry mouth, sweating, increased appetite, arthralgia, nausea, vomiting, headache, insomnia, difficult in micturition, nightmares, impotence, delayed ejaculation, rash, purpura, weight gain, hyperexcitability, increased neuromuscular activity and other extrapyramidal symptoms. Drug fever is extremely rare. Reduction in blood sugar and hypoglycemic effects are possible. Congestive heart failure has been reported in a few patients with reduced cardiac reserve.

Methyclothiazide: Blood dyscrasias (thrombocytopenia with purpura, agranulocytosis, aplastic anemia); elevation of BUN, blood sugar or serum uric acid (gout may be induced); anorexia, nausea, vomiting, diarrhea, headache, dizziness, paresthesia, weakness, skin rash, photosensitivity, jaundice and pancreatitis. Cutaneous vasculitis in elderly patients has been reported with other thiazides.

If side effects are severe or persist, reduce dosage or withdraw drug. 804439R

ABBOTT

TM-TRADEMARK

Harvard Medical School and Harvard School of Public Health, was Professor of Bacteriology at George Washington University from 1958 to 1960 and Professor of Microbiology and Community Health in 1960–61. He left George Washington to become Associate Professor, later Chairman of the Department, of Microbiology at Harvard School of Public Health. He came to Washington as Director of the FDA Bureau of Medicine in 1966.

PERSONALS

Dr. William B. Walsh, founder of Project HOPE and President of the People-to-People Health Foundation, was awarded an honorary degree, Doctor of Science with citation, at the graduating exercises of St. John's University in Jamaica, N. Y., June 9. Dr. Walsh was among 9 distinguished recipients representing the fields of education, music, medicine, poetry, humanitarianism, religion, government, science, and business so honored. They included Marianne Moore, Kate Smith, Archbishop Luigi Raimondi, Dr. René Jules Dubos, and others. The awards were conferred by University President, the Very Reverend Joseph T. Cahill, C.M.

Dr. Walsh is a graduate of St. John's College. His citation read in part:

> "Most men have sympathy for the miseries of their fellow men, far fewer have compassion for them, and it is the rare man indeed who feels them so deeply that he dedicates himself to their alleviation. Dr. William B. Walsh is such a man.
>
> ". . . Dr. Walsh was moved by the medical destitution of the islanders of the South Pacific to found Project HOPE . . . from his conviction that, although neither money nor staff enough could ever be found to combat disease effectively in a hundred far-flung communities, it was quite feasible to serve them with a floating medical center."

Dr. Thomas W. Mattingly was honored by the Republic of Tunisia at special ceremonies at the Blair House, May 17. President Habib Bourguiba conferred upon him the grade of Officer of the Order of the Republic in recognition of what Dr. Mattingly has accomplished "to strengthen the relations of friendship and cooperation between the Republic of Tunisia and the United States of America."

Dr. Clayton B. Ethridge, Professor Emeritus of Medicine at George Washington University, was presented the Certificate of Coöperation by the Department of State and the Agency for International Development, June 19. It was the first such award presented to an individual and was given to Dr. Ethridge for his assistance in providing training for foreign participants sponsored by A.I.D. The presentation took place at the 12th Annual Conference on Medical Education for Foreign Scholars in the Medical Sciences at Airlie House in Warrenton, Va.

Dr. James P. Mann has been elected President of the Potomac Tuberculosis and Respira-

DOCPAC BRIEFS

or

"My Political Creed"

DOCPAC (District of Columbia Political Action Committee) has received from AMPAC (AMA Political Action Committee) an attractively printed copy of the following Creed. If anyone would like to have a copy of this Creed, in color, suitable for framing, please write DOCPAC, 825 New Hampshire Avenue, N.W., Washington, D. C. 20037.

My Political Creed

I am a free citizen in a free nation.

Whoever diminishes my freedom as an individual diminishes the sum total of freedom in my country.

I am also a physician, free thus far to treat my patients to the best of my ability.

Abridge that freedom and the health of my patients is adversely affected.

These things being true, I cannot—I will not—stand idly by when these hard-won freedoms are under attack.

For I believe that the values upon which this country was founded are immutable and have not been eroded by the passage of years.

I believe that I, as a free citizen in a free land, am obligated to defend my beliefs in the ways permitted to me, and required of me, by our form of government.

Therefore, let no man seek to bar me from the political process; for it would be akin to denying my right to participate in the process that determines free government.

William H. Cooper, M.D.
Chairman, D.C. Political
Action Committee

tory Disease Association. He is Associate Clinical Professor of Medicine at George Washington University School of Medicine and founder and Director of the Clinic for emphysema patients in the Alexandria Community Health Center.

Dr. Monroe J. Romansky, Professor of Medicine at George Washington University School of Medicine, was invited to give the Annual Aaron Brown Lecture of Phi Delta Epsilon at the Medical College of Virginia in Richmond. The title of his address was "Factors Influencing the Trend in Antimicrobial Therapy."

►OBITUARIES

Adolphus Bogardus Bennett, M.D.

(1879–1968)

Dr. A. B. Bennett, Past President of the Medical Society of the District of Columbia and organizer of the United States Power Squadrons, died on June 9, 1968, at his home, 33 Kalorama Circle, N.W., at the age of 88.

Born at Brantford, Ontario, October 19, 1879, the son of Adolphus Bogardus Bennett and Susan Wilkes Bennett, he came with his parents at an early age to Washington, where he attended elementary schools in Georgetown, Western High School, and received his degree in medicine at Columbian (now George Washington) University in 1901. At Garfield Memorial Hospital he was an externe in 1899 and 1900 and an interne in 1901 and 1902.

He began general practice in Georgetown in 1902, having obtained his license in 1901. Postgraduate work at Harvard University under Mosher in 1904 and at Haale a/Saale, Germany, under Grünert in 1905 was followed by staff appointments in his specialty, otolaryngology, at Garfield Hospital, Episcopal Eye, Ear and Throat Hospital, and Columbia Hospital for Women. In April 1955, at Garfield, he was awarded the Goldheaded Cane for outstanding service to the Hospital.

He was commissioned First Lieutenant in the D. C. National Guard Medical Corps in 1901, and in March, 1913 was commissioned Lieutenant in the United States Naval Medical Reserve. He maintained offices in the old Farragut Medical Building from 1906 to 1926, and in the Washington Medical Building at 1801 Eye Street from 1926 to 1966, when he retired from practice.

He became President of the D. C. Medical Society in 1934; he had been an Active member since 1902 and a Life member since 1943.[1] He was Past President of the International Medical Club and of the George Washington University Medical Society and a diplomate of the American Board of Otolaryngology. The author of many papers in his specialty, he was the first to report on aural typhoid carriers.[2]

A. B. BENNETT, M.D.

Dr. Bennett was a life member of the Sons of the Revolution, a member of the Association of Oldest Inhabitants of the District of Columbia, and a member of the New York Avenue Presbyterian Church.

His chief and absorbing extracurricular activity was yachting. In 1914 he organized the Potomac River Power Squadron and was its Commander for 9 years. In 1919 he became Vice Commander of the United States Power Squadrons, made it an educational institution, and saved it from dissolution. He held the post of Chief Commander of the Squadrons for 4 years. He was a life member of the Corinthian Yacht Club and served as its Commodore for 10 years.

He is survived by his wife, Mrs. Ayleene Bennett, to whom he was married in 1954. He also leaves a sister, Mrs. Muriel Pfahler, of Philadelphia.

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[1] Ramsey, H. P.: Our Living Past Presidents. Med. Ann. D.C., 1950, 19, 699.

[2] Bennett, A. B.: Aural typhoid carriers: report of 2 cases. J.A.M.A., 1917 (January 6).

His first wife, Susan Beirne Patton, of Huntsville, Alabama, was a lineal descendant of Dr. Ephraim McDowell, of Danville, Kentucky, who successfully performed the first oöphorectomy (then called "ovariotomy") in the United States in 1809.

By the terms of the will of Dr. Bennett, his ashes were committed to the Potomac River he so much loved on June 17, the ceremony being performed from the deck of *Aragon II*, his last ship.

H. P. R.

James Hawfield, M.D.

(1893–1968)

Dr. James Hawfield, general practitioner and surgeon in the Washington area for nearly 50 years, died at Doctors Hospital, June 26, 1968. He lived at 2354 48th Street, N.W., and maintained offices at 1150 Connecticut Avenue, N.W.

Dr. Hawfield was born, July 2, 1893, in Monroe, North Carolina. He attended Duke University and the University of North Carolina for premedical study; he received his medical degree from Jefferson Medical College in Philadelphia in 1918. He interned for a year at Episcopal Hospital in Philadelphia and stayed on as resident physician for 2 more years.

He began private practice in the District of Columbia in 1921. He had staff privileges at Doctors, Sibley Memorial and Children's Hospitals, Columbia Hospital for Women, and the Washington Hospital Center.

Dr. Hawfield was an Active member of the Medical Society of the District of Columbia since 1925 and a Life member since 1965. He was a Fellow of the American College of Surgeons and a member of the American Medical Association and the Hipprocrates-Galen Medical Society. He was a member and Past President of the North Carolina State Society and the University of North Carolina Alumni Association. He was a member of the Metropolitan Memorial Methodist Church, the Masonic Lodge, and the Chevy Chase Club.

Survivors include his wife, Mrs. Anne Watson Hawfield; 2 sons, James H. Hawfield, of Bethesda, Md., and B. Mason Hawfield, of Littleton, N.C.; and a daughter, Mrs. James K. Foley, of Bethesda. He also leaves a sister, Mrs. Hampton Price, of Monroe, N.C., and 3 brothers, Dr. Clayton Hawfield, of Washington, and Roy R. and Frank Hawfield, of Monroe.

Philip Diatz, M.D.

(1900–1968)

Dr. Philip Diatz, anesthesiologist, died, June 14, 1968 at the Washington Hospital Center. He had been in ill health for several years.

A native of Lithuania, Dr. Diatz was born, May 14, 1900, and came to the United States in 1906. A graduate of old Central High School, he received his medical degree from George Washington University in 1926. He interned at the University and Children's Hospitals.

Dr. Diatz was in general practice for 10 years, after which he went to Bellevue Hospital in New York for postgraduate study in anesthesiology. He also trained in this specialty at Gallinger (now D. C. General) Hospital.

He was associate staff anesthesiologist at Columbia Hospital for Women and at Doctors Hospital and was affiliated in his specialty with Children's and Sibley Memorial Hospitals. From 1954 to 1958 he was Associate Chief of Anesthesiology at the old Hahnemann Hospital and was Chief of Anesthesiology at Hahnemann in 1958–59.

Dr. Diatz was elected to Active membership in the Medical Society of the District of Columbia in 1929. He was a member of the American Medical Association, the Southern Medical Association, the International Anesthesia Research Society, the American Society of Anesthesiologists, the Jacobi Medical Society, and the George Washington University Medical Alumni Association. He was a member of the B'nai B'rith Hebrew Congregation and served on its Board of Governors.

Surviving him are his wife, Mrs. Sonia Diatz, of 7520 16th Street, N.W.; a daughter, Mrs. Joy Renee Golden, of Silver Spring, Md.; and 2 sons, Richard S. Diatz, of Potomac, Md., and Sylvan L. Diatz, of Silver Spring, Md.

Shigellosis in District of Columbia—Hayman

(*Continued from page 422*)

except among family contacts. The incidence of Shigella is much higher in the District than in Maryland, Virginia, the United States as a whole, and New York City. This is explicable, at least in part, on the basis of the large indigent population in Washington and the pattern of their medical care. Most are treated at D. C. General Hospital or Children's Hospital where stool cultures are taken and results reported. The unusually high incidence of Shigella has continued during the first 2 months of 1968.

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helps restore normal motility and tone

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Diarrhea and other symptoms of common G.I. disorders can often be curbed with Cantil (mepenzolate bromide), bringing welcome relief to the harassed patient. Relatively specific for the hyperactive colon, it helps reduce diarrhea, pain and spasm with minimal effect on other viscera. Cantil (mepenzolate bromide) is indicated whenever these symptoms are associated with irritable colon, gastroenteritis, diverticulitis, and mild to moderate ulcerative colitis.

It is an anticholinergic drug without narcotic properties. Side effects are usually mild.

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CONTRAINDICATIONS: Sensitivity to Cantil® (mepenzolate bromide), glaucoma, G.I. or G.U. obstruction, toxic megacolon. WARNINGS: Possibility of adverse reactions in advanced severe ulcerative colitis and atypical agitated reactions to phenobarbital in the elderly (Cantil with Phenobarbital). PRECAUTIONS: Cantil is of adjunctive use only; treatment of the underlying condition is indicated, be it organic or functional. Observe caution in conditions known to be incompatible with atropine-like drugs, e.g., open-angle glaucoma or prostatic hypertrophy. ADVERSE EFFECTS: Dry mouth; blurred vision; constipation; nausea; vomiting; bloating; dizziness; urinary retention. All are anticholinergic effects and usually tolerable. Habituation and idiosyncratic reactions to phenobarbital (Cantil with Phenobarbital) are possible, as is generalized rash. DOSAGE: One or two tablets three times a day and one or two at bedtime usually provide prompt relief. Cantil with Phenobarbital may be prescribed if sedation is required. HOW SUPPLIED: CANTIL (mepenzolate bromide)—25 mg. per scored tablet. Bottles of 100 and 250. CANTIL with PHENOBARBITAL—containing in each scored tablet 16 mg. phenobarbital (warning: may be habit forming) and 25 mg. mepenzolate bromide. Bottles of 100 and 250.

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Don't let monilia cut broad-spectrum therapy short...

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tetracycline phosphate complex-nystatin

Use of broad-spectrum antibiotics can cause fungal overgrowth in the alimentary tract... and give rise to symptoms so troublesome that therapy must be prematurely stopped. Tetrex-F (tetracycline phosphate complex-nystatin) helps you circumvent this problem.

The nystatin can prevent overgrowth of monilia; the phosphate complex delivers tetracycline to the blood rapidly. Side effects are infrequent.

High-Risk Patients

Tetrex-F (tetracycline phosphate complex-nystatin) is especially useful in patients most susceptible to fungal overgrowth during tetracycline therapy: (1) the elderly or debilitated, (2) young children, (3) the diabetic, (4) those on long-term tetracycline therapy, (5) those on steroid therapy, (6) those who have had moniliasis before, and (7) pregnant patients with a history of monilial vaginitis.

When you start with economical Tetrex-F (tetracycline phosphate complex-nystatin), you can complete the full course of broad-spectrum therapy with less chance of losing control elsewhere. A good start for a healthy finish.

PRESCRIBING INFORMATION. For complete information consult Official Package Circular. *Indications:* Infections of respiratory, gastrointestinal and genitourinary tracts and skin and soft tissues due to tetracycline-sensitive organisms, in patients with increased susceptibility to monilial infections. *Contraindications:* The drug is contraindicated in patients hypersensitive to its components. *Warnings:* Photodynamic reactions have been produced by tetracyclines. Natural and artificial sunlight should be avoided during therapy. Stop treatment if skin discomfort occurs. With renal impairment, systemic accumulation and hepatotoxicity may occur. In this situation, lower doses should be used. Tooth staining and enamel hypoplasia may be induced during tooth development (last trimester of pregnancy, neonatal period and childhood). *Precautions:* Bacterial superinfections may occur. Infants may develop increased intracranial pressure with bulging fontanels. In gonorrheal therapy, serologic tests for syphilis should be conducted initially and monthly for 3 months. *Adverse Reactions:* Glossitis, stomatitis, nausea, diarrhea, flatulence, proctitis, vaginitis, dermatitis, and allergic reactions may occur. *Usual Adult Dosage:* 1 capsule q.i.d. Continue for 10 days in Beta-hemolytic streptococcal infections. Administer one hour before or two hours after meals. *Supplied:* Capsules, bottles of 16 and 100. Each capsule contains tetracycline phosphate complex equivalent to 250 mg. tetracycline HCl activity and 250,000 units of nystatin. For Oral Suspension, 125 mg. tetracycline and 125,000 u. nystatin/5 ml., 60 ml. bottles.

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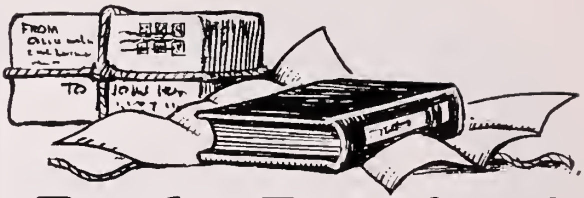


Books Received

ACKNOWLEDGMENT IS MADE of the following books which have recently been received.

Strabismus Ophthalmic Symposium (I). Edited by JAMES H. ALLEN, M.D. Price, $23.00. Pp. 476, with 181 illustrations. St. Louis: Mosby, 1950; second printing, 1968.

Organization and Management of Family Practice. Prepared by COMMITTEE ON MEDICAL ECONOMICS, A.A.G.P. Price (paper), $1.00. Pp. 105. Kansas City, Mo.: American Academy of General Practice, 1968.

The Care of the Geriatric Patient, 3rd ed. Edited by E. V. COWDRY, PH. D., SC.D. (Hon.). Price, $15.75. Pp. 430, with 19 illustrations. St. Louis: Mosby, 1968.

The First International Congress of Orthoptists. Transactions of a Congress held at the Royal College of Surgeons of England, July 3, 4, 5, 1967. Price, $14.25. Pp. 376. St. Louis: Mosby, 1968.

Ciba Foundation Symposium: Cell Differentiation. Edited by A. V. S. DEREUCK AND JULIE KNIGHT. Price, $12.00. Pp. 257, with 43 illustrations. Boston: Little, 1967.

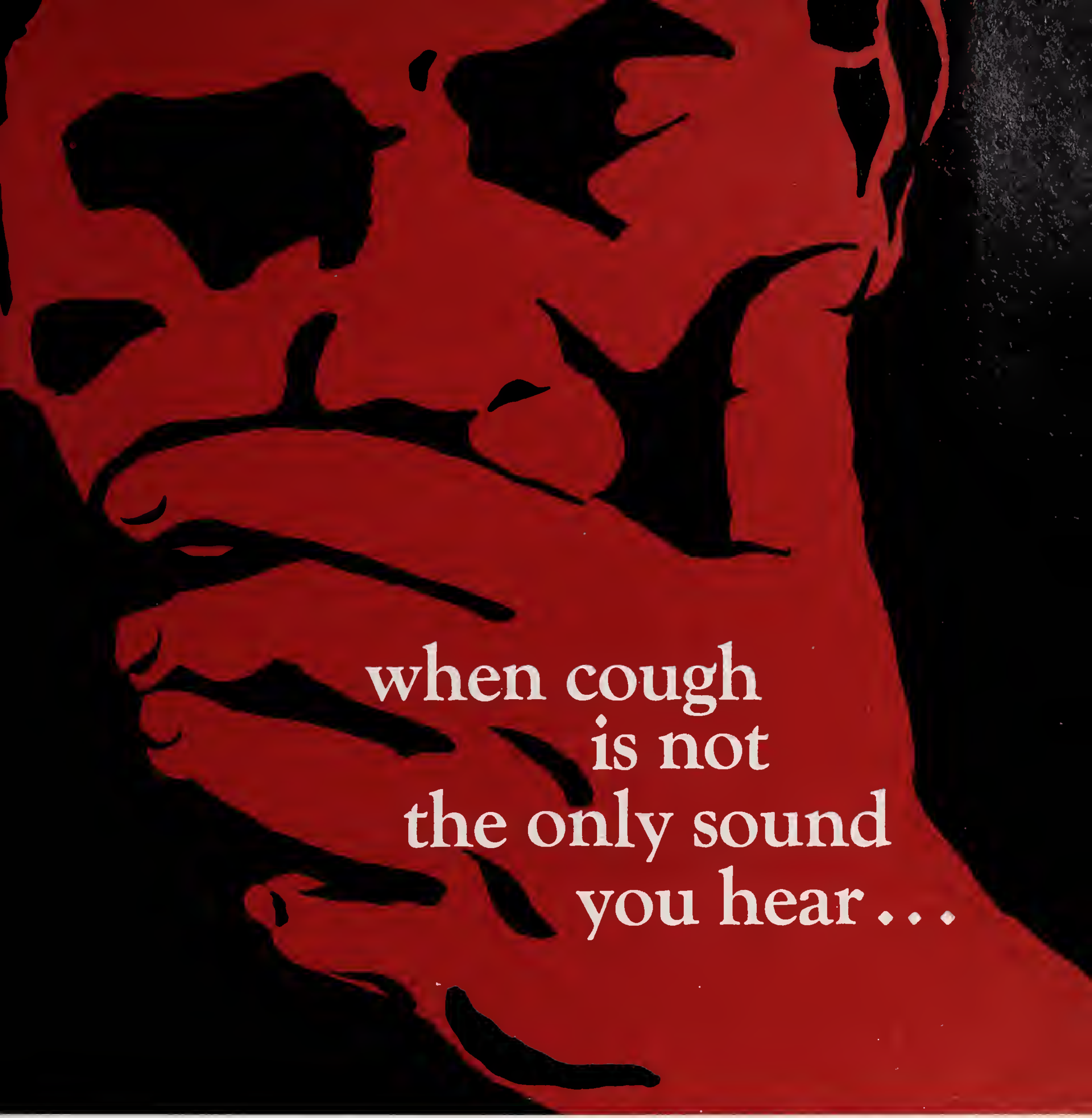
Ciba Foundation Volume. Communication in Science: Documentation and Automation. Edited by ANTHONY DEREUCK AND JULIE KNIGHT. Price, $12.50. Pp. 274, with 17 illustrations. Boston: Little, 1967.

Proceedings of the Second International Symposium on Plastic and Reconstructive Surgery of the Eye and Adnexa. BYRON SMITH, M.D., AND JOHN MARQUIS CONVERSE, M.D., Co-chairmen; DONALD WOOD-SMITH, F.R.C.S.E., AND MARGARET F. OBEAR, M.D., Secretaries; and 60 contributors. Price, $31.00. Pp. 548, with 303 illustrations. St. Louis: Mosby, 1967.

U. S. Department of Health, Education and Welfare: Report of the National Conference on Medical Costs, held in Washington, D. C., June 27–28, 1967. Price (paper), $1.25. Pp. 326. Washington, D. C. 20402: U. S. Government Printing Office, 1968.

Ciba Foundation Study Group No. 30: The Balkan Nephropathy. Edited by G. E. W. WOLSTENHOLME AND JULIE KNIGHT. Price, $3.50. Pp. 123, with 29 illustrations. Boston: Little, 1967.

Ciba Foundation 100th Symposium: Health of Mankind. Edited by GORDON WOLSTENHOLME AND MAEVE O'CONNOR. Price, $12.00. Pp. 297, with 38 illustrations. Boston: Little, 1967.



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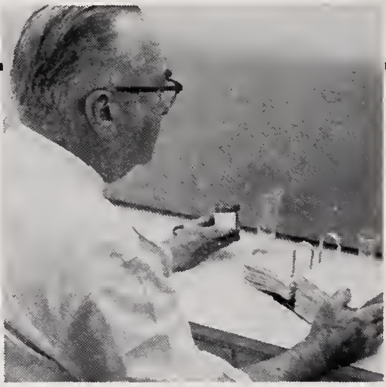

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Adult Immunization Guide. A working manual for doctors and other health personnel, the booklet describes immunization procedures for some 19 infectious diseases. 55 pp. American College Health Association, Box 9117, University of Miami Branch, Coral Gables, Fla. 33124.

Classification and Management of Diabetes Mellitus. Arthur Krosnick, M.D. Lists atypical signs of diabetes which might first be seen by any of 16 specialists. Free copies available from Diabetes Detection Program, 342 Madison Avenue, New York, N. Y. 10017.

Directory of Homemaker-Home Health Aide Services (1966–67). Lists over 800 agencies providing homemaker services in the United States and Canada. Price, $3.00. National Council for Homemaker Services, 1740 Broadway, New York, N. Y. 10019.

Fads, Myths, Quacks—and Your Health (Public Affairs Pamphlet No. 415). Jacqueline Seaver. 28 pp. Price, 25 cents. Public Affairs Committee, 381 Park Avenue South, New York, N. Y. 10016.

Federal Benefits for Veterans and Dependents. New legislative changes including the Veterans' Pension and Readjustment Assistance Act of 1967 and reviews all existing benefits available for veterans. Price, 20 cents. Superintendent of Documents, U. S. Government Printing Office, Washington, D. C. 20402.

The Merck Index, 8th ed. Paul G. Stecher and Merck staff. Encyclopedia of chemicals and drugs; offers descriptive monographs of over 9,500 chemicals and drugs arranged alphabetically by generic or nonproprietary name. Cross Index of Names, containing 42,000 entries, enables readers to locate chemical descriptions by chemical-generic or proprietary name. Price, $15.00. Merck & Co., Inc., Publications Department, Rahway, N. J. 07065.

A Source Book on Food Practices, with Emphasis on Children and Adolescents. Miss E. Beyer. Information on the food practices of infants, children, and adolescents. 19 pp. National Dairy Council, 111 North Canal Street, Chicago, Ill. 60606.

Theological Roots for a Hospital Ministry. VA Chaplain Workshops 1966 (VA No. 10-85). Robert E. Hunt, Leo Jung and Albert L. Meiburg. Three historical and incisive papers by theologians of the Catholic, Jewish, and Protestant faiths. 38 pp. Price, 20 cents. Superintendent of Documents, U. S. Government Printing Office, Washington, D. C. 20402.

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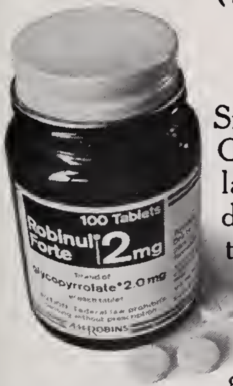

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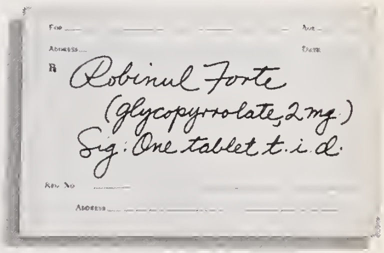

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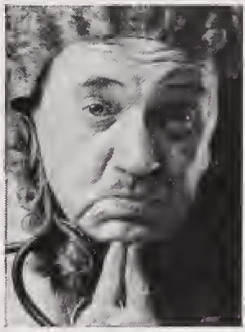

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Contraindications: Known hypersensitivity to drug; children under 6 months of age; acute narrow angle glaucoma; may be used in patients with open angle glaucoma who are receiving appropriate therapy.

Warnings: Not of value in treatment of psychotic patients, and should not be employed in lieu of appropriate treatment. As with most CNS-acting drugs, caution patients against hazardous occupations requiring complete mental alertness (*e.g.,* operating machinery, driving). When used adjunctively in convulsive disorders, possibility of increase in frequency and/or severity of grand mal seizures may require increase in dosage of standard anticonvulsant medication; abrupt withdrawal in such cases may also be associated with temporary increase in frequency and/or severity of seizures. Advise patients against simultaneous ingestion of alcohol and other CNS depressants. Withdrawal symptoms (similar to those with barbiturates and alcohol) have occurred following abrupt discontinuance. Keep addiction-prone individuals (such as drug addicts or alcoholics) under careful surveillance because of their predisposition to habituation and dependence. Use of any drug in pregnancy, lactation or in women of childbearing age requires that potential benefit be weighed against possible hazard.

Precautions: If combined with other psychotropics or anticonvulsants, carefully consider individual pharmacologic effects—particularly with known compounds which may potentiate action of Valium (diazepam), such as phenothiazines, narcotics, barbiturates, MAO inhibitors and other antidepressants. Employ usual precautions in the severely depressed or in those with latent depression; suicidal tendencies may be present and protective measures necessary. Observe usual precautions in impaired renal or hepatic function. Limit dosage to smallest effective amount in elderly and debilitated to preclude ataxia or oversedation (initially 2 to 2½ mg once or twice daily, increasing gradually as needed or tolerated).

Adverse Reactions: Side effects most commonly reported: drowsiness, fatigue and ataxia. Infrequently encountered: confusion, constipation, depression, diplopia, dysarthria, headache, hypotension, incontinence, jaundice, changes in libido, nausea, changes in salivation, skin rash, slurred speech, tremor, urinary retention, vertigo and blurred vision. Paradoxical reactions such as acute hyperexcited states, anxiety, hallucinations, increased muscle spasticity, insomnia, rage, sleep disturbances and stimulation have been reported; should these occur, use of the drug should be discontinued. Because of isolated reports of neutropenia and jaundice, periodic blood counts and liver function tests are advisable during long-term therapy. Minor changes in EEG patterns (low-voltage fast activity) observed during and after therapy and are of no known significance.

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Muscle Pain Following Succinylcholine Chloride
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Extradural and Subdural Empyemas
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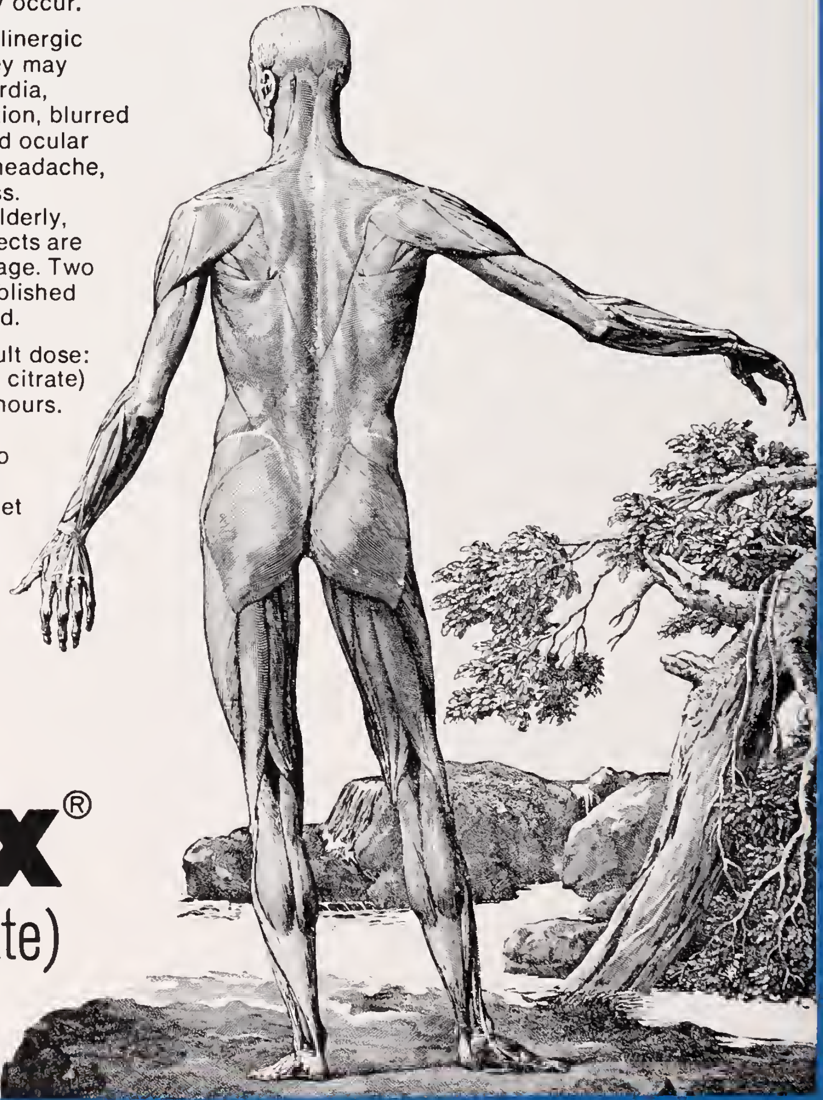

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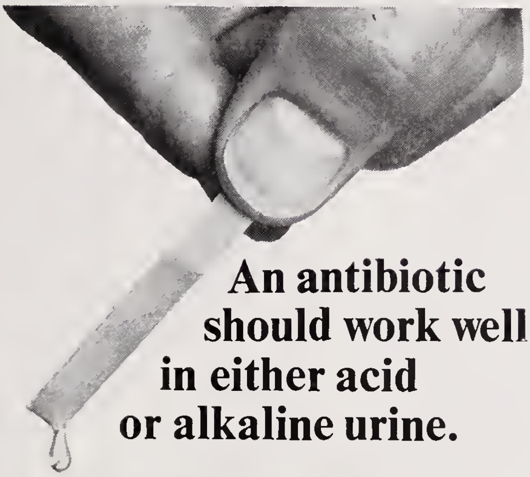

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Contraindication: History of hypersensitivity to demethylchlortetracycline.

Warning: In renal impairment, usual doses may lead to excessive accumulation and liver toxicity. Under such conditions, lower than usual doses are indicated, and, if therapy is prolonged, serum level determinations may be advisable. A photodynamic reaction to natural or artificial sunlight has been observed. Small amounts of drug and short exposure may produce an exaggerated sunburn reaction which may range from erythema to severe skin manifestations. In a smaller proportion, photoallergic reactions have been reported. Patients should avoid direct exposure to sunlight and discontinue drug at the first evidence of skin discomfort. Necessary subsequent courses of treatment with tetracyclines should be carefully observed.

Precautions: Overgrowth of nonsusceptible organisms may occur. Constant observation is essential. If new infections appear, appropriate measures should be taken. In infants, increased intracranial pressure with bulging fontanels has been observed. All signs and symptoms have disappeared rapidly upon cessation of treatment.

Side Effects: Gastrointestinal system – anorexia, nausea, vomiting, diarrhea, stomatitis, glossitis, enterocolitis, pruritus ani. Skin – maculopapular and erythematous rashes; a rare case of exfoliative dermatitis has been reported. Photosensitivity; onycholysis and discoloration of the nails (rare). Kidney – rise in BUN, apparently dose-related. Transient increase in urinary output, sometimes accompanied by thirst (rare). Hypersensitivity reactions–urticaria, angioneurotic edema, anaphylaxis. Teeth – dental staining (yellow-brown) in children of mothers given this drug during the latter half of pregnancy, and in children given the drug during the neonatal period, infancy and early childhood. Enamel hypoplasia has been seen in a few children. If adverse reaction or idiosyncrasy occurs, discontinue medication and institute appropriate therapy. Demethylchlortetracycline may form a stable calcium complex in any bone-forming tissue with no serious harmful effects reported thus far in humans.

Average Adult Daily Dosage: 150 mg q.i.d. or 300 mg b.i.d. Should be given 1 hour before or 2 hours after meals, since absorption is impaired by the concomitant administration of high calcium content drugs, foods and some dairy products. Treatment of streptococcal infections should continue for 10 days, even though symptoms have subsided.

Capsules: 150 mg; Tablets: film coated – 300 mg, 150 mg and 75 mg of demethylchlortetracycline HCl.

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Tandearil® in Osteoarthritis
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Contraindications: Edema; danger of cardiac decompensation; history or symptoms of peptic ulcer; renal, hepatic or cardiac damage; history of drug allergy; history of blood dyscrasia. The drug should not be given when the patient is senile or when other potent drugs are given concurrently.
Warning: Tandearil is an analog of phenylbutazone; sensitive patients may be cross-reactive. If coumarin-type anticoagulants are given simultaneously, watch for excessive increase in prothrombin time. Instances of severe bleeding have occurred. Persistent or severe dyspepsia may indicate peptic ulcer; perform upper gastrointestinal x-ray diagnostic tests if drug is continued. Pyrazole compounds may potentiate the pharmacologic action of sulfonylurea, sulfonamide-type agents and insulin. Carefully observe patients receiving such therapy. Use with caution in the first trimester of pregnancy and in patients with thyroid disease.
Precautions: Before prescribing, carefully select patients, avoiding those responsive to routine measures as well as contraindicated patients. Obtain a detailed history and a complete physical and laboratory examination, including a blood count. The patient should not exceed recommended dosage, should be closely supervised and should be warned to discontinue the drug and report immediately if fever, sore throat, or mouth lesions (symptoms of blood dyscrasia), sudden weight gain (water retention), skin reactions, black or tarry stools or other evidence of intestinal hemorrhage occur. Make complete blood counts at weekly intervals during early therapy and at 2-week intervals thereafter. Discontinue the drug immediately and institute countermeasures if the white count changes significantly, granulocytes decrease, or immature forms appear. Use greater care in the elderly and in hypertensives.
Adverse Reactions: The more common are nausea and edema. Swelling of the ankles or face may be minimized by withholding dietary salt, reduction in dosage or use of diuretics. In elderly patients and in those with hypertension, the drug should be discontinued with the appearance of edema. The drug has been associated with peptic ulcer and may reacti-

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e a latent peptic ulcer. The patient should
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als or with milk to minimize gastric upset.
ug rash occasionally occurs. If it does,
mptly discontinue the drug. Agranulocy-
is, exfoliative dermatitis, Stevens-Johnson
drome, Lyell's syndrome (toxic necrotiz-
epidermolysis) or a generalized allergic
ction similar to a serum sickness syn-
me may occur and require permanent
hdrawal of medication. Agranulocytosis
occur suddenly in spite of regular, re-
ated normal white counts. Stomatitis, sali-
y gland enlargement, vomiting, vertigo and
guor may occur. Leukemia and leukemoid
ctions have been reported. While not defi-
ely attributable to the drug, a causal rela-
ship cannot be excluded. Thrombocytopenic purpura and aplastic anemia may occur. Confusional states, agitation, headache, blurred vision, optic neuritis and transient hearing loss have been reported, as have hyperglycemia, hepatitis, jaundice, hypersensitivity, angiitis, pericarditis and several cases of anuria and hematuria. With long-term use, reversible thyroid hyperplasia may occur infrequently. Moderate lowering of the red cell count due to hemodilution may occur.

For complete details, please see full Prescribing Information.

Dosage in Osteoarthritis: Initial: 3 to 6 tablets daily in divided doses. Usually unnecessary to exceed 4 tablets daily. A trial period of one week is considered adequate to determine the therapeutic effect of the drug. Maintenance: Effective level often achieved with 1 or 2 tablets daily, should not exceed 4 tablets daily. In selecting appropriate dosage in any specific case, consideration should be given to the patient's weight, general health, age and other factors influencing drug response.
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October 4–8: International Congress on Diseases of the Chest. Washington Hilton Hotel, Washington. Address: American College of Chest Physicians, 112 East Chestnut Street, Chicago, Ill. 60611.

October 10–12: American Thyroid Association. Hotel Statler-Hilton, Washington. Address: Dr. William M. McConahey, Executive Secretary, Mayo Clinic, Rochester, Minn. 55901.

October 12: American College of Physicians (Maryland/D. C. Regional). Georgetown University School of Medicine, Washington. Address: Dr. Thomas W. Mattingly, 2007 Eye Street, N.W., Washington, D. C. 20006.

October 12–18: College of American Pathologists. Fontainebleau Hotel, Miami Beach, Fla. Address: Mr. Oliver J. Neibel, Jr., Executive Director, 230 North Michigan Avenue, Chicago, Ill. 60601.

October 13–16: Medical Society of Virginia. Hotel Roanoke, Roanoke, Va. Address: Mr. Robert I. Howard, Executive Secretary, 4205 Dover Road, Richmond, Va. 23221.

October 14–18: American College of Surgeons. Dennis Hotel, Atlantic City, N. J. Address: Dr. John Paul North, 55 East Erie Street, Chicago, Ill. 60611.

October 19–23: American Society of Anesthesiologists. Washington Hilton Hotel, Washington. Address: Mr. J. W. Andes, Executive Secretary, 515 Busse Highway, Park Ridge, Ill. 60068.

October 19–24: American Academy of Pediatrics. Palmer House, Chicago. Address: Dr. Robert G. Frazier, Executive Director, 1801 Hinman Avenue, Evanston, Ill. 60204.

October 20–23: Association of Military Surgeons of the United States. Sheraton Park Hotel, Washington. Address: Brig. General Frank E. Wilson, USAR (Ret.), 1500 Massachusetts Avenue, N.W., Washington, D. C. 20005.

October 27: American Association of Ophthalmology. Palmer House, Chicago. Address: Mr. Lawrence A. Zupan, Executive Secretary, 1100 17th Street, N.W., Washington, D. C. 20036.

October 28–31: American Association of Blood Banks. Shoreham Hotel, Washington. Address: Miss Lois J. James, Executive Secretary, 30 North Michigan Avenue, Chicago, Ill. 60602.

November 21–23: Medical Society of the District of Columbia. Washington Hilton Hotel, Washington. Address: Mr. Thomas Sadler, Executive Director, 2007 Eye Street, N.W., Washington, D. C. 20006.

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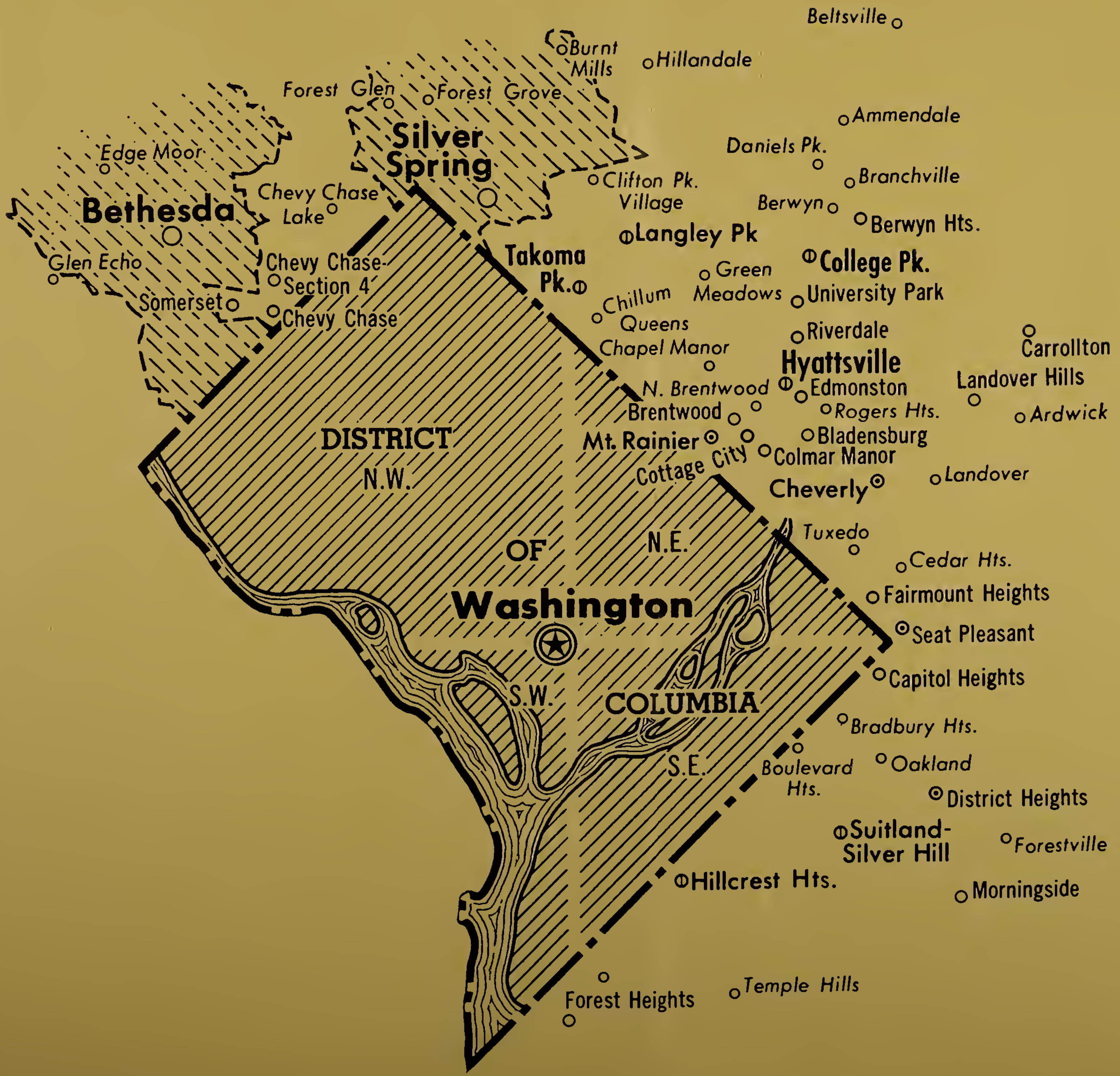

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Precautions: Use may result in overgrowth of nonsusceptible organisms. Constant observation is essential. If new infections appear, take appropriate measures. Use of tetracycline during teeth development may cause discoloration of teeth.

Side Effects: Gastrointestinal system—anorexia, nausea, vomiting, diarrhea, stomatitis, glossitis, enterocolitis, pruritus ani. Skin—maculopapular and erythematous rashes (a case of exfoliative dermatitis has been reported); photosensitivity reaction, onycholysis and discoloration of nails (rare). Kidney—rise in BUN, apparently dose-related. Hypersensitivity reactions—urticaria, angioneurotic edema, anaphylaxis. In young infants, bulging fontanels have been reported following full therapeutic dosage. This symptom has disappeared rapidly when drug is discontinued. Teeth—dental staining (yellow-brown) in children of mothers given tetracycline during the latter half of pregnancy, and in children given the drug during the neonatal period, infancy and early childhood. Enamel hypoplasia has been seen in a few children. Blood—anemia, thrombocytopenic purpura, neutropenia, eosinophilia. Liver—cholestasis (rare), usually at high dosage. Tetracycline may form a stable calcium complex in bone-forming tissue. If adverse reaction or idiosyncrasy occurs, discontinue medication and institute appropriate therapy.

Average Adult Daily Dosage: One Gm. per day, in 4 divided doses of 250 mg. each. Should be given 1 hour before or 2 hours after meals, since absorption is impaired by the concomitant administration of high calcium content drugs, foods and some dairy products. Treatment of streptococcal infections should continue for 10 days, even though symptoms have subsided.

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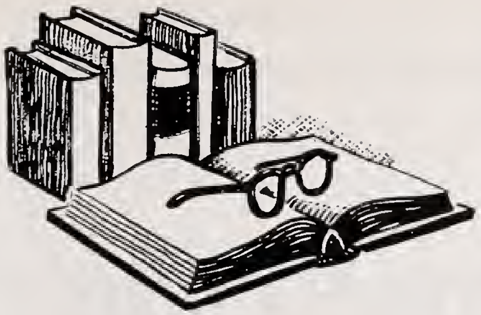


Book Reviews

Medical Department, United States Army: Neuropsychiatry in World War II. Vol. 1, Zone of Interior. COLONEL ROBERT S. ANDERSON, Editor in Chief; COLONEL ALBERT J. GLASS AND LIEUTENANT COLONEL ROBERT J. BERNUCCI, Editors for Neuropsychiatry. Prepared and published under the direction of Lieutenant General Leonard D. Heaton, The Surgeon General, United States Army. Price, $7.50. Pp. 898, with 65 illustrations. Washington, D. C. 20402: U. S. Government Printing Office, 1967.

This volume is essentially the comprehensive story of psychiatry in World War II, with a chapter devoted to neurology. In it is recorded with detail and authenticity "the administrative and professional frustrations, failures, and successes as they occurred in the Zone of the Interior." An official compilation of this history was anticipated long before the termination of hostilities. Related materials and documents were accumulated and stored by responsible members of the Surgeon General's Office and the staff of the Neuropsychiatry Consultants Division. Key personnel and many of the assigned consultants were periodically brought into the division to record their experiences before they were deployed elsewhere or released from the Army. For a period of 12 years attempts by part-time historians to produce this history were fruitless. It was only after a full-time project officer was assigned and an Advisory Editorial Board was appointed that the task was accomplished. The final effort is the result of the contributions of 70 authors, co-authors, and collaborators. Many of the latter have been and are currently leaders in American psychiatry.

The editors have moulded voluminous material into a comprehensive, encyclopedic, yet well-organized and readable history. Statistics are readily available for any aspect of the period yet do not detract from the story.

The story contains:

1. A brief résumé of psychiatry in World War I.
2. Contrasting features of the 2 wars.
3. The application of lessons derived from World War I to World War II and how many of these proved inappropriate.
4. The relationship of manpower effectiveness to emotional and personality disorders.
5. The development of effective procedures for the prevention and management of psychiatric problems in soldiers.
6. The demonstration of the effectiveness of good leadership and morale in reducing psychiatric casualties.

A valuable addition to this history is a final chapter, "Lessons Learned." A summary of the lessons are:

1. Failure of responsible military authorities during mobilization and early phases of hostilities to appreciate the inevitability of large-scale psychiatric disorders under conditions of modern warfare. In combat alone a ratio of 1 psychiatric casualty for every 4 battle casualties has prevailed regularly since World War I.
2. Military psychiatric leadership in the Office of the Surgeon General must be operational during peacetime and should not be discarded for reasons of expediency or economy.
3. Psychiatric screening was unable to identify effectively and thus eliminate the military psychiatric problem at induction. Psychiatric screening as performed in World War II was an impractical and ineffective procedure.
4. For the purely symptomatic disorders and maladjustment problems, hospitalization itself created or perpetuated illness and disability.
5. Consultation services proved effective in preventing and reducing psychiatric casualties.
6. Psychiatric diagnosis influenced the disposition of marginal military personnel. The diagnosis of "psychoneurosis" was abused and contributed to a "gain in illness." A revision in nomenclature to include the abundant transient personality reactions and immaturity reactions has enabled the military psychiatrist to better evaluate the problem and procure a more adequate disposition.

SEYMOUR J. ROSENBERG, M.D.

Textbook of Anatomy and Physiology in Radiologic Technology. CHARLES A. JACOBI, B.Sc., R.T. (A.R.R.T.). Price, $12.00. Pp. 421, with 187 illustrations. St. Louis: Mosby, 1968.

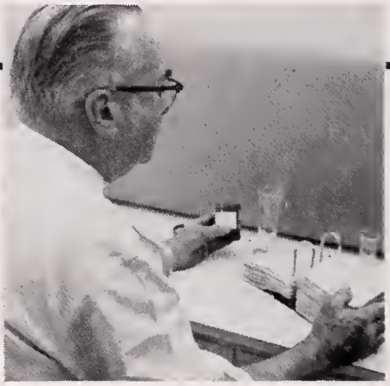
This volume is the result of an ambitious project to include within one cover most of the anatomy and physiology background which a radiologic technologist should need in his regular and special duties. The book is organized into an introductory chapter, a chapter on Basic Body Structures and Functions, an embryologic survey, and detailed descriptive chapters on each of the 9 major organ systems.

The early chapters are comprehensive and contain some condensed material, such as terminology, anatomic location and direction, which probably needs expansion for the benefit of the beginning student. Some material, as the physiology of membranes and

(Continued on page 17)

Count the Prescriptions filled by Peoples Drug Stores and you get 100 million times people have counted on Peoples

We have passed the hundred million mark. That's the number of prescriptions filled by all Peoples Drug Stores since we opened our doors in 1905. We say this with great pride, since this impressive figure illustrates an impressive public confidence. Because behind all prescribed medicine at Peoples is confidence — in the physician who prescribes, the manufacturer who supplies, and the pharmacist who fills the prescription. At Peoples, nothing is more important to us than this confidence.



PEOPLES
PRESCRIPTION DRUG STORES

(*Continued from page 15*)

fluid and electrolyte balance, would need considerable class explanation to a student without an adequate background of elementary chemistry. One may question the inclusion of the Embryologic Survey, with its necessary specialized terminology, as a subject to be mastered by the student technologist, except in special interests where the need of such knowledge is indicated. The chapters on the organ systems make excellent use of anatomic models as well as high quality radiographs as illustrations. Assembly of such material could be better arranged in some places, as in the correlations between models, radiographs, and subject photographs of the various positions of the heart in Chapter 6. The book has a usable Glossary of Prefixes, Suffixes and Terms, and it is well indexed.

While this text probably needs supplementation by more elementary illustrative material and visual aids for the average beginning student of radiologic technology, it may serve as an excellent reference source for practicing technologists and teachers of radiologic technology. It is perhaps of limited usefulness to radiologists and other physicians.

FREDERICK D. DREW, M.D.

The Heart and the Law. E. L. SAGALL, M.D., AND B. C. REED, LL.B. Price, $28.00. Pp. 842, with 42 illustrations. New York: Macmillan, 1968.

The book constitutes the most extensive and complete publication under one cover on the subject of medicolegal cardiology. However, if the lawyer believes that the knowledge in this book will make him a cardiologist or the cardiologist believes that the information contained herein will make him a lawyer, they are both in error. Rather than accomplishing such a task, this book does provide a comprehensive and scholarly reference to the physician and the attorney as well as to many other individuals who become involved with the problem of determining the relationship of physical and psychic trauma to primary injury to the normal heart or to the precipitation or aggravation of preëxisting disease in an abnormal heart by trauma and physical effort. Most of all, it should improve the communication and understanding among physicians, lawyers, claims investigators, and others involved in the adjudication of claims relative to disease and trauma to the heart.

Contained in its 48 chapters and 5 appendices is a tremendous collection of the information now available on this subject, including such current subjects as cardiac arrest, untoward effects of drugs, diagnostic procedures, etc. Appendix V consists of the reproduction of "Guides to the Evaluation of Cardiac Disability" as prepared by the Committee on Medical Rating of Physical Impairment of the American Medical Association, of which the late George W.

(*Continued on page 19*)

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painful night
leg cramps...
respond to specific
therapy with
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BRISTOL

BRISTOL LABORATORIES
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Syracuse, New York 13201

Book Reviews

(*Continued from page 17*)

Cooley was Secretary. Each chapter is supported with footnotes listing appropriate medical and legal references, including legal case citations and additional source material recommended for the reader.

An excellent comprehensive subject index of 24 pages greatly facilitates the utilization of this text. A short medical glossary is contained in the appendix which will provide assistance to the nonmedical reader but, unfortunately, the physician is not provided with a glossary for assistance in interpreting the common but confusing legal and insurance terms.

This book treats rather extensively the subject of cardiac disability as settled out of the court room but as translated into damages, awards, and benefits under workmen's compensation, insurance policies, and retirement programs.

The price of $28 for this book may appear excessive to the casual user or reader of such a text but should be no deterrent to the individual who is working in the field of medicolegal cardiology. It certainly should be available as a reference in the libraries of hospitals, cardiac clinics, and other institutions concerned with cardiac injury and disease.

THOMAS W. MATTINGLY, M.D.

Practical Automation for the Clinical Laboratory. WILMA L. WHITE, B. A., MARILYN M. ERICKSON, B.S., AND SUE C. STEVENS, B.A., M.A., PH.D. Price, $14.50. Pp. 401, with 242 illustrations. St. Louis: Mosby, 1968.

This book attempts to give laboratory personnel a fairly thorough basic understanding of the mechanics and electronics involved in the use of automative equipment. A chapter is devoted to the principles of mechanics and electronics of machines, in general, and a glossary of terms is provided. The specific mechanics, operation, methods of maintenance and trouble shooting of the autoanalyzer, atomic absorption, and electrophoretic instruments are dealt with in detail. Other chapters discuss the Mecolab, the Robot Chemist, the ultra autotechnicon and data-processing instrumentation. Warner-Chilcott and the Technicon Corporation have contributed significantly to the book. Excellent diagrams and photographs have been incorporated in the text.

Although this book probably has a relatively limited circulation potential, it should prove of very significant value to laboratory directors and to those individuals charged with the understanding, preventive maintenance, and trouble shooting of a progressively greater variety of instruments. It should prove of value to instructors in medical technology schools who might use the text for required supplemental student reading.

MARY J. MATTHEWS, M.D.

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Parepectolin for quick relief of acute diarrhea
...soothes colicky pain with paregoric*
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and protects intestinal mucosa

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Each fluid ounce of creamy white suspension contains:
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Contains opium (¼ grain) 15 mg. per fluid ounce.
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Pectin .... (2½ grains) 162 mg.
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In addition to its primary indications for duodenal and gastric ulcer, Robinul Forte (glycopyrrolate) is indicated for other G-I conditions that may benefit from anticholinergic therapy. Robinul-PH Forte (glycopyrrolate 2 mg. with phenobarbital) is indicated when these situations are complicated by mild anxiety and tension.

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Precautions: Administer with caution in the presence of incipient glaucoma.

Adverse Reactions: Dryness of the mouth, blurred vision, urinary difficulties, and constipation are rarely troublesome and may generally be controlled by reduction of dosage. Other side effects associated with the use of anticholinergic drugs include tachycardia, palpitation, dilatation of the pupil, increased ocular tension, weakness, nausea, vomiting, headache, dizziness, drowsiness, and rash.

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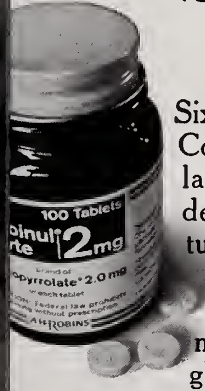
In peptic ulcer therapy, won't you ive Robinul® Forte a Fair Trial? (glycopyrrolate)



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wever, we believe you'll agree there's always m for a better anticholinergic. This is why we're ing you to give Robinul Forte a fair trial. Robinul te exerts a highly specific antisecretory action and ked inhibitory effect on intestinal tone. We're con-ed you'll agree that this is indeed an outstanding g when you observe its outstanding suppression of r symptoms. Furthermore, it is unique in that it uces intestinal tone, yet has little or no effect on istalsis. In addition, the incidence of the more hersome peripheral side effects is low.

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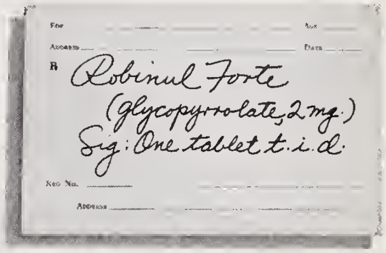
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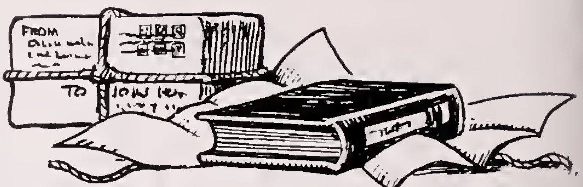

Books Received

Textbook of Anatomy and Physiology in Radiologic Technology. CHARLES A. JACOBI, B.SC., R.T. (A.R.R.T.). Price, $12.00. Pp. 421, with 187 illustrations. St. Louis: Mosby, 1968.

Dyslexia: Diagnosis and Treatment of Reading Disorders. Edited by ARTHUR H. KEENEY, M.D., D.SC., AND VIRGINIA T. KEENEY, M.D. Price, $12.00. Pp. 182, with 24 illustrations. St. Louis: Mosby, 1968.

Infectious Diseases of Children, 4th ed. SAUL KRUGMAN, M.D., AND ROBERT WARD, M.D. Price, $16.50. Pp. 428, with 69 figures and 9 color plates (including a gatefold insert). St. Louis: Mosby, 1968.

Medical Physiology, 12th ed. Volumes I and II. Edited by VERNON B. MOUNTCASTLE, M.D. Price per 2-volume set, $24.00. Pp. 1054 (Vol. I.), 1055–1858 (Vol. II), with 880 illustrations, including 1 in color. St. Louis: Mosby, 1968.

Decision Making in the National Science Policy: A Ciba Foundation and Science Foundation Symposium. Edited by ANTHONY DE REUCK, MAURICE GOLDSMITH, AND JULIE KNIGHT. Price, $12.00. Pp. 310. Boston: Little, 1968.

Pediatric Therapy, 3rd ed. HARRY C. SHIRKEY, B.S. (PHARM.), M.D., F.A.A.P., with 89 contributors. Price, $25.00. Pp. 1294, with 351 illustrations. St. Louis: Mosby, 1968.

Anesthesia for Infants and Children, 3rd ed. ROBERT M. SMITH, M.D. Price, $17.50. Pp. 530, with 240 illustrations. St. Louis: Mosby, 1968.

Principles of Nuclear Medicine. Edited by HENRY N. WAGNER, JR., M.D. Price, $27.50. Pp. 896, illustrated. Philadelphia: Saunders, 1968.

Practical Automation for the Clinical Laboratory. WILMA L. WHITE, B.A., MARILYN M. ERICKSON, B.S., AND SUE C. STEVENS, B.A., M.A., PH.D. Price, $14.50. Pp.401, with 242 illustrations. St. Louis: Mosby, 1968.

Ciba Foundation Symposium: Interferon. Edited by G. E. W. WOLSTENHOLME AND MAEVE O'CONNOR. Price, $12.00. Pp. 284, with 54 illustrations. Boston: Little, 1967.

Ciba Foundation Study Group No. 31: Nutrition and Infection. Edited by G. E. W. WOLSTENHOLME AND MAEVE O'CONNOR. Price, $3.50. Pp. 144, with 20 illustrations. Boston: Little, 1968.

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Warning: In renal impairment, since liver toxicity is possible, lower doses are indicated; during prolonged therapy consider serum level determinations. Photodynamic reaction to sunlight may occur in hypersensitive persons. Photosensitive individuals should avoid exposure; discontinue treatment if skin discomfort occurs.

Precautions: Drowsiness, anorexia, slight gastric distress can occur. In excessive drowsiness, consider longer dosage intervals. Persons on full dosage should not operate vehicles. Nonsusceptible organisms may overgrow; treat superinfection appropriately. Treat beta-hemolytic streptococcal infections at least 10 days to help prevent rheumatic fever or acute glomerulonephritis. Tetracycline may form a stable calcium complex in bone-forming tissue and may cause dental staining during tooth development (last half of pregnancy, neonatal period, infancy, early childhood).

Adverse Reactions: *Gastrointestinal*—anorexia, nausea, vomiting, diarrhea, stomatitis, glossitis, enterocolitis, pruritus ani. *Skin*—maculopapular and erythematous rashes; exfoliative dermatitis; photosensitivity; onycholysis, nail discoloration. *Kidney*—dose-related rise in BUN. *Hypersensitivity* reactions—urticaria, angioneurotic edema, anaphylaxis. *Intracranial*—bulging fontanels in young infants. *Teeth*—yellow-brown staining; enamel hypoplasia. *Blood*—anemia, thrombocytopenic purpura, neutropenia, eosinophilia. *Liver*—cholestasis at high dosage.

Upon adverse reaction, stop medication and treat appropriately.

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349-8

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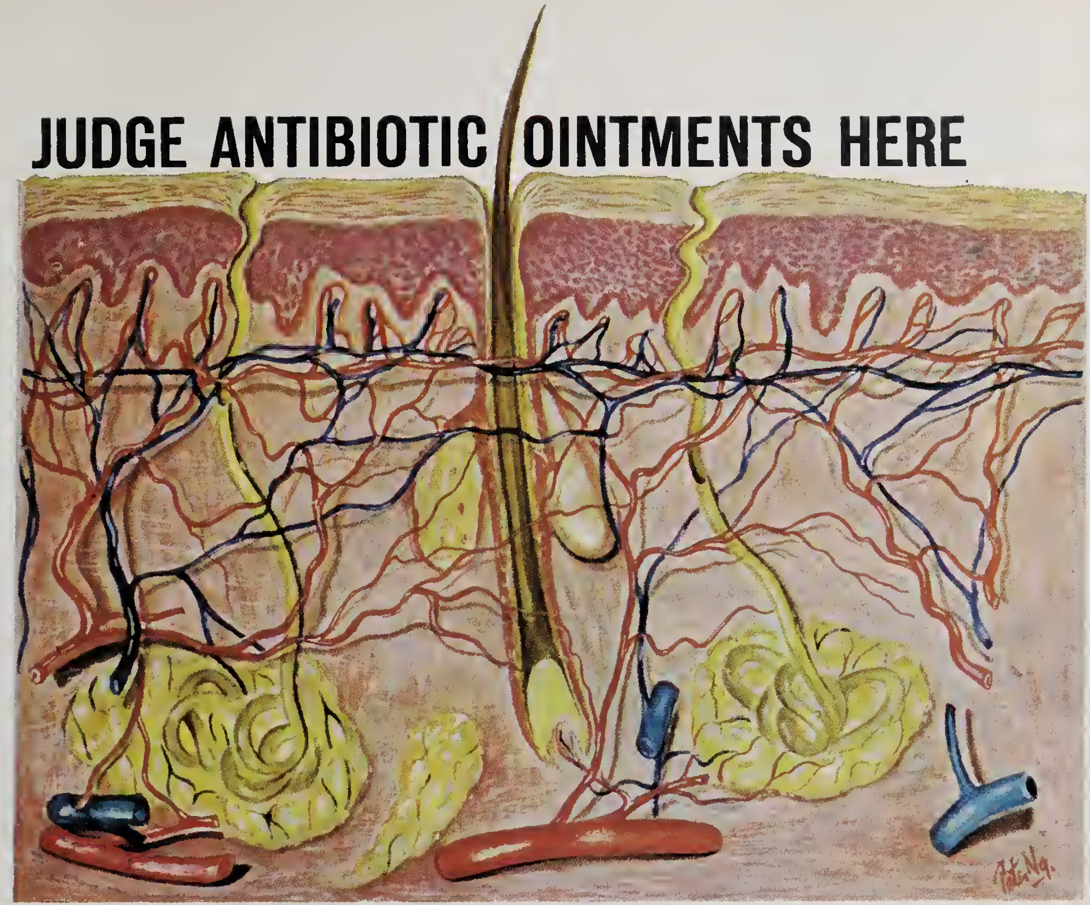

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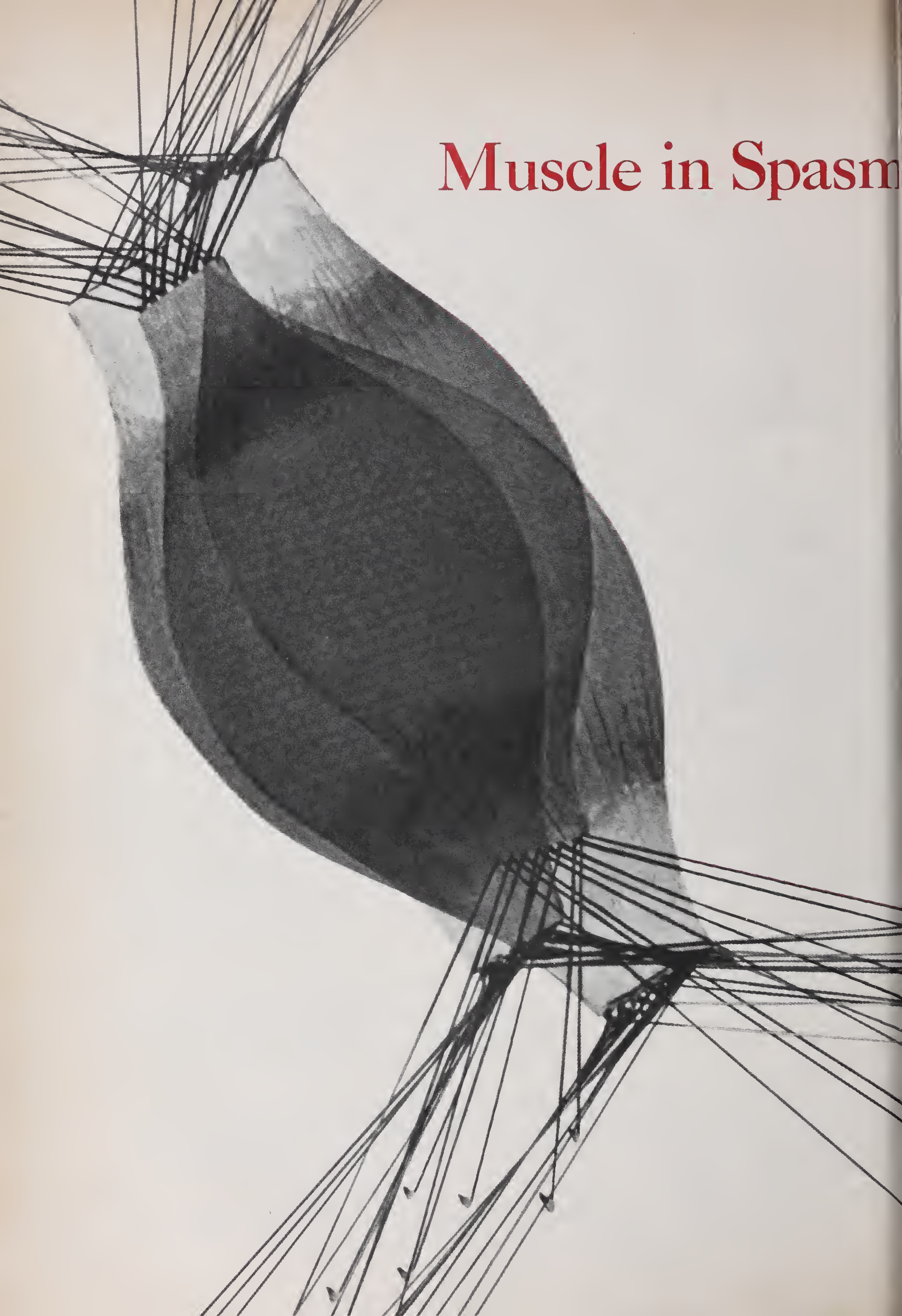

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The ability of Valium to help relieve skeletal muscle spasm—as well as psychic tension—demonstrates its clinical value and versatility.

The muscle-relaxant effect obtained with Valium, used adjunctively with other drugs or physiotherapy, favorably affects the entire cluster of spasm-related symptoms . . . helps accelerate return to normal activity.

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Indications: Tension and anxiety states; somatic complaints which are concomitants of emotional factors; psychoneurotic states manifested by tension, anxiety, apprehension, fatigue, depressive symptoms or agitation; acute agitation, tremor, delirium tremens and hallucinosis due to acute alcohol withdrawal; adjunctively in skeletal muscle spasm due to reflex spasm to local pathology, spasticity caused by upper motor neuron disorders, athetosis, stiff-man syndrome, convulsive disorders (not for sole therapy).

Contraindicated: Known hypersensitivity to the drug. Children under 6 months of age. Acute narrow angle glaucoma.

Warnings: Not of value in psychotic patients. Caution against hazardous occupations requiring complete mental alertness. When used adjunctively in convulsive disorders, possibility of increase in frequency and/or severity of grand mal seizures may require increased dosage of standard anticonvulsant medication; abrupt withdrawal may be associated with temporary increase in frequency and/or severity of seizures. Advise against simultaneous ingestion of alcohol and other CNS depressants. Withdrawal symptoms have occurred following abrupt discontinuance. Keep addiction-prone individuals under careful surveillance because of their predisposition to habituation and dependence. In pregnancy, lactation or women of childbearing age, weigh potential benefit against possible hazard.

Precautions: If combined with other psychotropics or anticonvulsants, consider carefully pharmacology of agents employed. Usual precautions indicated in patients severely depressed, or with latent depression, or with suicidal tendencies. Observe usual precautions in impaired renal or hepatic function. Limit dosage to smallest effective amount in elderly and debilitated to preclude ataxia or oversedation.

Side Effects: Drowsiness, confusion, diplopia, hypotension, changes in libido, nausea, fatigue, depression, dysarthria, jaundice, skin rash, ataxia, constipation, headache, incontinence, changes in salivation, slurred speech, tremor, vertigo, urinary retention, blurred vision. Paradoxical reactions such as acute hyperexcited states, anxiety, hallucinations, increased muscle spasticity, insomnia, rage, sleep disturbances, stimulation, have been reported; should these occur, discontinue drug. Isolated reports of neutropenia, jaundice; periodic blood counts and liver function tests advisable during long-term therapy.

Valium® (diazepam)
2-mg, 5-mg, 10-mg tablets

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Louie lost weeks with
a painful shoulder. That's a lot of fish.
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If it doesn't work in a week, forget

But please don't forget this:

Contraindications: Edema; danger of cardiac decompensation; history or symptoms of peptic ulcer; renal, hepatic or cardiac damage; history of drug allergy; history of blood dyscrasia. The drug should not be given when the patient is senile or when other potent drugs are given concurrently. Large doses of the alka formulation are contraindicated in glaucoma.

Warning: If coumarin-type anticoagulants are given simultaneously, watch for excessive increase in prothrombin time. Instances of severe bleeding have occurred. Persistent or severe dyspepsia may indicate peptic ulcer; perform upper gastrointestinal x-ray diagnostic tests if drug is continued. Pyrazole compounds may potentiate the pharmacologic action of sulfonylurea, sulfonamide-type agents and insulin. Carefully observe patients receiving such therapy. Use with caution in the first trimester of pregnancy and in patients with thyroid disease.

Precautions: Before prescribing, carefully select patients, avoiding those responsive to routine measures as well as contraindicated patients. Obtain a detailed history and a complete physical and laboratory examination, including a blood count. The patient should not exceed recommended dosage, should be closely supervised and should be warned to discontinue the drug and report immediately if fever, sore throat, or mouth lesions (symptoms of blood dyscrasia); sudden weight gain (water retention); skin reactions; black or tarry stools or other evidence of intestinal hemorrhage occur. Make complete blood counts at weekly intervals during early therapy and at 2-week intervals thereafter. Discontinue the drug immediately and institute countermeasures if the white count changes significantly, granulocytes decrease, or immature forms appear. Use greater care in the elderly and in hypertensives.

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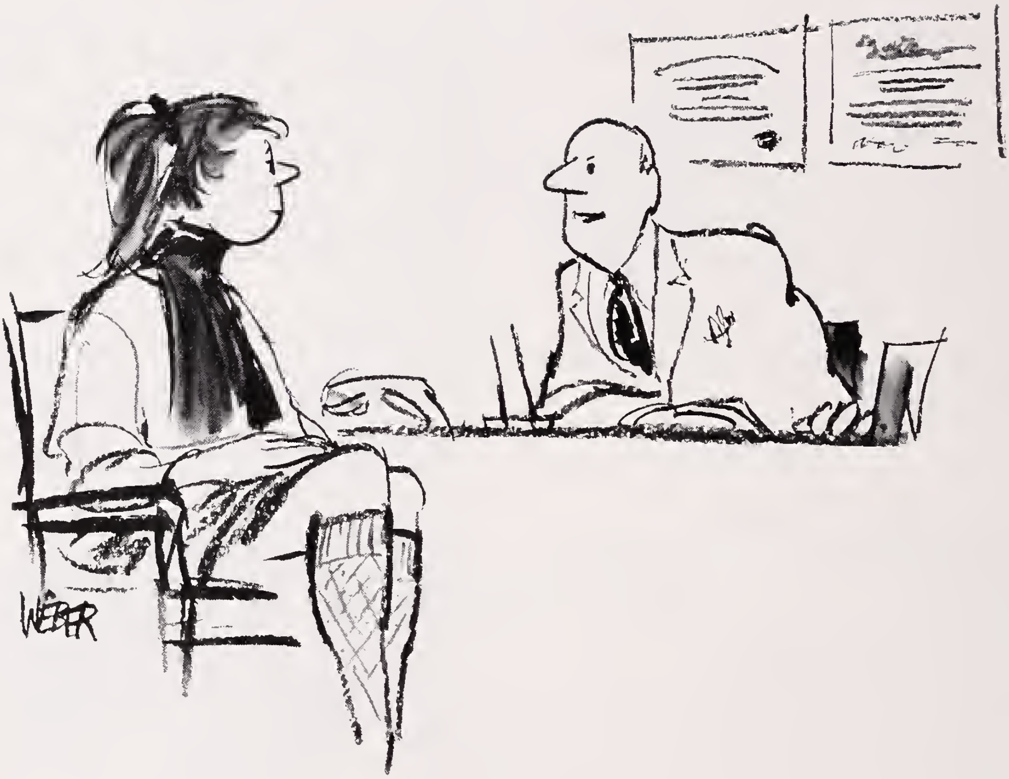

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Washington Hospital Center Number

This is the sixth of a fifth series of special numbers of the Medical Annals, the scientific material for which is supplied by medical institutions and scientific organizations in the Washington Metropolitan Area. For the Washington Hospital Center Number the Editorial Board is indebted to Dr. Lawrence E. Putnam, Chairman of the Editorial Committee of the Center, who secured the papers which appear in this issue.

Many excellent papers were submitted for this issue but due to lack of space some of them have been held over for later publication.

Management of Infected Arterial Prostheses

A Review of Current Methods and a New Experimental Technique

PHILIP J. FERRIS, M.D.,* AND CSABA L. MAGASSY, M. D.†

Synthetic materials have provided excellent grafts for the vascular surgeon for replacement of arterial segments. They are generally strong, durable, inert, readily available, easily stored, and technically easily adaptable. When infected, however, they become a nidus for harboring microorganisms with invariable catastrophic result to life or limb.

Infection of arterial prostheses is generally considered to be uncommon, although Healey et al[1] report a 5 per cent incidence of bacterial infection in aortic grafts. Surprisingly, Boyd and McCann[2] found 20 positive cultures in 71 abdominal aortic aneurysms. In instances of major trauma in which bowel and major arteries are involved, the need for an arterial replacement may create a serious dilemma because of contamination of the area in which the prosthesis must be placed.[3] In situations of spontaneous rupture of abdominal aneurysms into hollow viscera creating aorto-enteric fistulae, infection again constitutes the most distressing problem.[4, 5, 6] Mycotic aneurysms have also taxed the resources of the surgeon without clear course of action apparent. The eventual outcome of an infected aneurysm is fatal rupture; only resection can effect a cure, since antibiotics cannot sterilize these lesions.[5, 7, 8] In all of these situations, regardless of the ingenious methods used, results have been uniformly unacceptable and often catastrophic. Only in a few cases has a fatal outcome been averted.

Two major methods of management of infected arterial prostheses are proposed in reviewing the literature. The first method to be considered is based upon the theory that complete resolution of the infection can be accomplished if all devitalized tissue is completely débrided and appropriate antibiotics carried to all sites of bacterial growth.[9] Carter et al[10] reported satisfactory results in such situations with local wound débridement, instillation of topical antibiotics, and vigorous systemic antibiotic therapy while leaving the synthetic graft in place. Harrison[11] utilized similar principles in experimental animals with quite satisfactory results. Schramel and Creech[12] used similar methods in 6 patients, but 5 eventually required removal of the synthetic graft with resultant loss of extremity or death from complications of the infected graft.

Such experiences of morbidity and mortality have caused most authors to state that synthetic prostheses once infected must be totally removed.[9, 13, 14] Humphries[4] reviewed 70 cases of postoperative aorto-enteric fistulae and concluded that despite the possible danger of the loss of limb, all prostheses should be removed and the ends of the aorta oversewn. He thought that replacement by another graft in the presence of infection inevitably resulted in re-rupture within a short time. This conclusion was also similarly expressed by Crawford et al.[15] Bennett and Cherry[16] reported 6 cases of mycotic aneurysms that were resected and prostheses inserted. All patients died, thereby illustrating the hazards of recurrent infections and vascular disruption following grafting procedures in infected fields.

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If this latter method of management is attempted with removal of infected prosthesis to allow the infection to resolve, necessarily continuity of arterial blood flow is disrupted. Ingenious and complex bypass grafting procedures have been designed to allow the removing of the infected graft while delivering the necessary blood via a noncontaminated route. Blaisdell[13] proposed thoracic aorta-femoral artery bypass or axillary artery-femoral artery bypass[14] to provide lower extremity circulation when an infected abdominal aortic prosthesis was removed. Veith et al[6] proposed an upper abdominal aorta-femoral artery bypass via the rectus sheath for an infected aorto-iliac graft. Similar methods of exclusion have been reported by other authors.[17, 18] These procedures necessarily increase the extent of dissection and constitute almost insurmountable hazard when superimposed upon a preëxisting critical situation of sepsis.

Ideally, an alternative to either of these methods would combine certain features of both, i.e., allow removal of the synthetic graft and direct replacement with suitable material to withstand local infection and allow the resolution of the infection by ordinary therapeutic means. Necessarily, a satisfactory replacement tissue would have to have certain properties: ready accessibility, strength to withstand arterial pressure, durability to insure function at least until infection could be resolved, and a satisfactory host tolerance without serving as a nidus to harbor infection.

To satisfy the last criterion in particular, autogenous tissues were considered to hold the most promise. Venous autografts were acknowledged to satisfy all criteria most closely, but requirements of length and diameter might preclude their use in the eventuality of need for replacement of large arterial conduits such as the aorta or any of its major branches. Experimentally, a host of other autogenous tissues have been used for arterial reconstruction, including pericardium,[19] fascia,[20] dermis,[21] and skin,[22] all with less than acceptable results. Matsumoto et al[23, 24] reported satisfactory use of inverted segments of small bowel for replacement of large veins, but certainly hydrostatic pressure within venous channels is much lower than would have to be withstood in arterial replacement.

Experimental Study

Under aseptic conditions 16 mongrel dogs underwent resections of segments of their abdominal aortae. Details of technique are reported elsewhere.[25] Five of these animals had arterial continuity reëstablished by insertion of a free segment of inverted autogenous jejunum. The serosa was thereby rendered the inner lining of the graft. The mucosa was simply removed from the exterior of the graft by gently scraping with a scalpel. The segments of jejunum were invariably larger than the segments of aorta to be replaced; therefore, tailoring of the graft was performed by excising a longitudinal strip of the jejunum and suturing as necessary with 5-0 Tevdek®. In these 5 animals the inverted jejunal autograft was aseptically inserted after soaking in a 10 per cent neomycin solution for 15 to 20 minutes.

In the remaining 11 dogs, resection of segments of abdominal aorta was performed and initially replaced with Dacron® prostheses in standard fashion. These were then intentionally contaminated by direct application of 1 to 2 c.c. of a suspension of canine feces. The retroperitoneal areas were then closed and the peritoneal cavities irrigated with saline solution and neomycin solution in an attempt to confine the infection to the area of the graft. When the animals unquestionably showed evidence of clinical sepsis, usually 4 to 6 days later, they were reëxplored and the infected Dacron grafts removed. Inverted jejunal autografts were directly inserted as in the 5 dogs previously described (figure 1).

In the aseptic group 1 animal developed distemper and was sacrificed at 14 days. A small mural thrombus was adherent to the suture line where the graft had been tailored. This was about one-third the diameter of the lumen, but the graft was otherwise in good order. The remaining animals in this group were allowed to survive, and aortography was performed at 6 months. All grafts were functioning well then although 1 showed slight tortuosity, 2 showed minimal dilatation, and 1 showed moderate dilatation. One of these animals was then sacrificed for histologic studies, and the remaining 3 were followed for 1

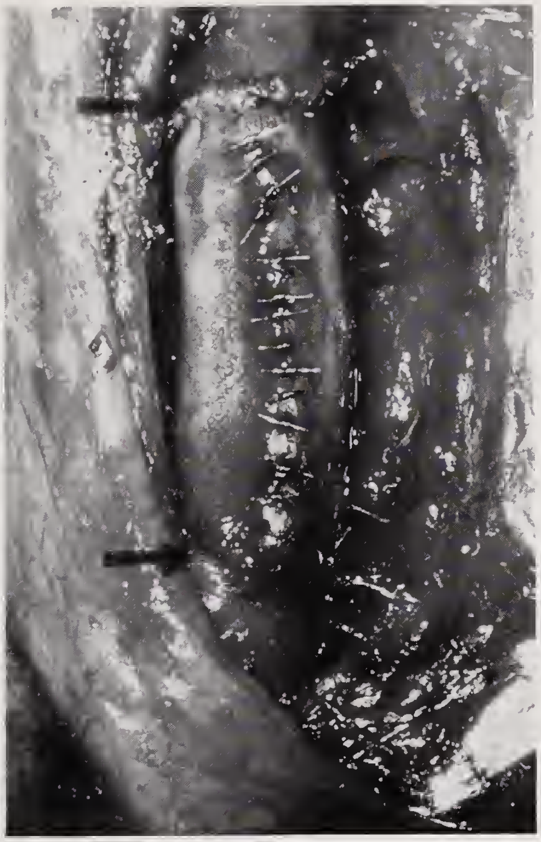

FIG. 1. Appearance of free inverted jejunal autograft immediately after insertion in an infected bed. Note the interrupted wire sutures where graft was tailored to appropriate caliber. Arrows indicate the anastomoses.

year. Aortography was then repeated, and significantly there was no progression of the dilatation of the grafts (figure 2).

In the intentionally infected group of dogs, not surprisingly 3 dogs died within 72 hours from the overwhelming sepsis. At autopsy the grafts, however, were in good order. Eight animals survived the initial septic process of which monofilament wire suture material was used in 7 for anastomoses and tailoring of the jejunal grafts. The eighth dog was the initial animal in this infected group, and Tevdek was used for suturing. This animal died at 5 days from disruption of the proximal suture line, at which point the adjacent aorta was necrotic. Since we considered the Tevdek to be not unlike other synthetic material in harboring infection, the use of monofilament 5-0 wire was instituted. In these 7 animals 1 died at 17 days from definite graft failure secondary to rupture of a focal area of necrosis. The jejunal autograft was otherwise intact. The remaining 6 dogs were allowed to survive 6 months, when aortograms showed moderate dilatation in 1, minimal dilatation in 2, and no dilatation or other abnormality in 3. All grafts were functioning well regarding the maintenance of arterial continuity.

These animals received Chloromycetin® therapy for approximately 2 weeks postoperatively in usual therapeutic dosages. Intravenous balanced electrolyte solutions or dextran were used for 2 to 3 days until the animals were able to sustain themselves by normal alimentation. Certainly no unusual measures were utilized regarding drugs in dosage or duration of administration.

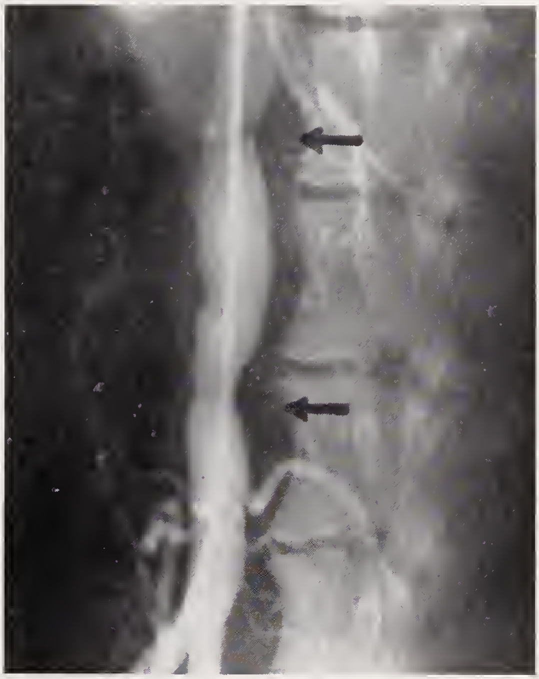

FIG. 2. Retrograde aortogram showing jejunal autograft 1 year after insertion for arterial replacements. Retrograde catheter is evident within the lumen. Arrows indicate the anastomoses.

Two animals were sacrificed at 6 months for histologic study. Two are presently alive and well at 9 and 11 months since operation respectively. Repeated aortograms and histologic studies are planned when they reach 1 year. In the 2 remaining animals, at 6 months reëxploration was performed and Dacron prostheses reinserted. Cultures in these animals showed no growth although previously coliform microörganisms had routinely been present. These animals are now 4 months since insertion of Dacron prostheses without evidence of recurrent infection or other apparent untoward results.

Histologic studies are reported in greater detail elsewhere,[25] but in summary the free jejunal grafts in both the aseptic and infected groups only seemed to vary in degree of surrounding inflammatory tissue and fibrosis. Pseudovasa vasorum were evident as early as 17 days after sacrifice with apparent parasitic type blood supply obtained from the adjacent retroperitoneal structures. The serosal layer remained as a smooth layer of endothial-like cells without evidence of lack in continuity. The longitudinal muscle showed progressive fibrosis, but the circular muscle seemed to remain well preserved. Submucosa was densely fibrotic in all specimens at 6 months and formed a sheath on the exterior of the graft resembling a fibrous adventitia.

Conclusions

Admittedly, our experimental groups of animals are small; however, certain significant features are evident. First, jejunal autografts are capable of withstanding the unrelenting arterial blood pressures while maintaining continuity of arterial flow for prolonged periods of time up to 1 year. Second, in the event of infection of synthetic arterial prostheses an alternate approach to the diametrically opposed, presently used methods of management is presented. The ability of inverted free jejunal autografts to survive in 75 per cent (6 out of 8) of our infected animals combines the more attractive features of both of these methods, which include removal of the infected prosthesis, direct reëstablishment of the vascular continuity, and resolution of the septic process by usual therapeutic measures. Third, in the situation of jejunal graft malfunction from any cause, the prosthetic graft can be reinserted after 6 months without recurrent infection.

No clinical experience has yet been obtained with the experimental method described. A study of this size would not usually be considered an adequate basis for clinical application; however, the usual therapeutic measures have consistently been so futile that this new technique is thought to be worthy of more consideration in the proper clinical situation.

Our sincere appreciation to Dr. Richard L. Levin for preparation of tissues for histologic study and their interpretation. The entire experimental study was supported by the Washington Hospital Center Surgical Research Fund.

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Ventilatory Disturbances Resulting from Muscle Pain Following the Administration of Succinylcholine Chloride*

JO SHIBUYA, M. D.,† NEMESIO CUEVO, M. D.,‡ FRED C. QUARNSTROM, D. D. S.,§
AND SOLOMON N. ALBERT, M. D.||

In recent years several reports have appeared on the incidence of muscle pain following the administration of succinylcholine chloride (SCC). Different methods to prevent this complication have been proposed, but none have proven effective in all instances. One cannot predict whether a patient will or will not develop this painful condition which usually appears within 24 hours following the administration of SCC.

Common complaints are a feeling of fatigue, "washed-out," "every muscle hurts," pain in the neck and back muscles, tightness of the chest, and inability to take a deep breath. The complaint that attracted our attention was the feeling of tightness of the chest and inability to take a deep breath because of pain. For this purpose a clinical study on ventilatory changes associated with post-succinylcholine chloride myalgia was conducted. This report presents our observations on 96 female patients undergoing minor surgery under general anesthesia, comparing the results of ventilatory changes that occur following the administration of SCC.

Method and Material

Ninety-six female patients ranging in age from 19 to 60 years of age in good physical condition admitted for a diagnostic dilation and curettage were selected for this study. The patients were seen the night before, and control studies were performed which consisted of:

1. Blood pH and blood gas analysis pCO_2, pO_2. These determinations were performed on arterialized blood drawn in a capillary tube from an earlobe puncture which was previously warmed and rubbed for 1 minute.[1, 2]
2. Ventilatory measurements, minute volume and tidal volume were performed with a Wright spirometer.
3. Maximal voluntary respiration (MVR). Patients were asked to breathe as deeply as possible over a 15-second period; the volume of air exchanged was measured during this period with a Wright spirometer.
4. Breathing reserve ratio was calculated according to the following equation:

$$\frac{\text{MVR} - \text{Minute Volume}}{\text{MVR}} \times 100$$

Preoperative medication consisted of 0.3–0.5 mg. atropine sulfate and 50–75 mg. meperidine hydrochloride administered intramuscularly 1 hour before surgery. General anesthesia consisted of the intravenous administration of sodium thiopental, 200–300 mg., supplemented by nitrous oxide and oxygen gas mixture, 2:2 liters per minute with 1–2 per cent halothane delivered by a Fluotec® vaporizer.

The patients were divided into 3 groups:

Group I. No SCC during the course of anesthesia.

Group II. 40–50 mg. SCC intravenously in a single dose after induction of anesthesia.

Group III. Gallamine triethiodide, 40 mg., intravenously followed by 40 mg. SCC.

Respiration was spontaneous in Group I and was manually assisted during the period of apnea in Groups II and III. Postoperatively the patients were allowed to ambulate at will. They were kept without sedation for 24 hours, at which time experimental studies were conducted.

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Results

Table 1 summarizes the incidence of myalgia as well as the extent and distribution of muscle pain. In Group I, of 25 patients (receiving no SCC) only 1 patient (4 per cent) complained of myalgia in the region of the neck. In Group II, of the 66 patients receiving SCC, 25 (37.8 per cent) complained of myalgia. The distribution and extent of myalgia are reported in table 1. In Group III, of the 5 patients receiving a dose of gallamine triethiodide prior to the administration of SCC there were no complaints of myalgia.

TABLE 1: Distribution and Incidence of Myalgia under General Anesthesia

Group	Total No. Cases	No. Cases with Postoperative Myalgia	Fatigue ("washed-out")	Pain in Extremities	Neck Pain	Chest Pain	Back Pain
Control	25	1	0	0	1	0	0
SCC	66	25	7*	5*	3†	2	10
SCC and gallamine	5	0	0	0	0	0	0

* Included 1 patient who complained of general malaise and back pain.

† Included 1 patient who complained of neck and back pain.

There were no significant changes in tidal and minute volume studies between the control group and the other 2 groups that received SCC and gallamine triethiodide followed by SCC. This was reflected in blood pH and pCO_2 and pO_2 analyses.

Table 2 summarizes the changes observed in respiratory patterns, especially concerned with MVR.

TABLE 2: Mean Postoperative Change of Minute Volume, Tidal Volume and Maximal Voluntary Respiration*

	No. Patients	Minute Volume (MV) Per cent	Tidal Volume (TV) Per cent	Maximal Voluntary Respiration (MVR) Per cent	Breathing Reserve Ratio (BRR) Per cent
Control	25	111.0	96.3	108.0	101.2
SCC without myalgia	41	95.7	91.2	85.8	91.2
SCC with myalgia other than back, chest pain	13	96.6	89.8	82.8	90.5
SCC with back, chest pain	12	97.2	91.4	59.0	72.0
SCC with gallamine	5	94.0	89.1	100.2	97.9

* Minute volume, tidal volume, MVR (maximal voluntary respiration) over 15 seconds. Breathing reserve ratio calculated from MVR and MV according to equation: (MVR − MV)/MVR × 100.

Discussion

The muscle pain following the administration of succinylcholine chloride is a painful and distressing condition. It was first reported by Bourne et al.[3] Churchill-Davidson[4] reported an incidence of 60 per cent, Hegarty[5] an incidence of 20–25 per cent, Morris and Dunn[6] an incidence of 6 per cent, and Leatherdale et al[7] 36 per cent. In a study conducted at the Washington Hospital Center[8] following vaginal delivery with the administration of SCC, the incidence of myalgia was 78 per cent in the SCC series versus 22 per cent in the control group. The incidence of myalgia in the present study is lower than the figures reported by the various investigators and even our own. The frequency, extent and duration of myalgia seem to vary with the extent of surgery, sex, age, and early ambulation,[9] and possibly other factors,[10] unknown at the present time, may also contribute to post-SCC myalgia. From our studies age did not seem to contribute to the incidence and distribution of painful muscles. Churchill-Davidson[11] implied that early ambulation may play an important role in the degree of myalgia. This also we did not notice in our cases.

The only finding of significance in this study was voluntary deep breathing and a reduction in maximal voluntary respiration. This ventilation test was introduced by Hermaneu in 1933[12] and was designed to measure the speed and efficiency of filling and emptying the lungs by increased ventilatory effort.

Significant changes were observed in the maximal voluntary respiration. No changes were seen from preoperative levels in both the control group and the group of 5 patients that received a dose of gallamine triethiodide prior to the administration of SCC. There was significant reduction in MVR in the group that received SCC, with a drop in MVR to 85.8 per cent of preoperative level in patients with no myalgia and a drop in MVR to 82.8 per cent of preoperative level in patients complaining of fatigue and general aches and pains. A most significant drop in MVR to 59 per cent of preoperative level was observed in the patients complaining of chest and back pain.

The breathing reserve ratio declined markedly in the group receiving SCC, and in one case there was a drop to 63.4 per cent of normal. Although dyspnea has been reported to appear when the breathing reserve ratio drops to 70 per cent of normal,[13] none of our patients had dyspnea despite the fact that this ratio had dropped significantly in a few instances and the patients felt tightness around the chest.

Summary and Conclusion

A study was conducted to determine the effect of muscle pain following the administration of succinylcholine chloride on ventilatory patterns. The only significant changes noted were a reduction in maximal voluntary respiration and the breathing reserve ratio. This seems to be directly related to the extent and distribution of painful muscles and is due to the fact that deep breathing is painful. The inability to breathe deeply postoperatively following the administration of SCC can contribute to the morbidity of patients and in particular the elderly and weak patient.

Any attempt to reduce or eliminate the incidence of post-succinylcholine chloride myalgia would seem to be of value. One possibility is the administration of a small dose of neuromuscular competitive blocking agent prior to the administration of SCC, a depolarizing agent.[4, 5, 14-20]

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Clinical Studies in the Use of Oral Diazoxide in Postsurgical and Functional Hypoglycemia*

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DIAZOXIDE is now recognized as a valuable drug for the treatment of patients with nonresectable, insulin-secreting tumors and in children with leucine-sensitive hypoglycemia.[1, 2, 3] While there is general agreement that the principal action of diazoxide is direct inhibition of insulin secretion by the beta-cell,[4] it is still believed that there is also a secondary extrapancreatic action, most likely on the adrenal medulla. However, the evidence for this second effect can be demonstrated only in animals.[5, 6]

In order to determine whether diazoxide would be useful in other hypoglycemic conditions we have studied the effect of this drug in patients with hypoglycemia secondary to gastric surgery and in others diagnosed as having "functional hypoglycemia."

Methods

All patients were admitted to the Clinical Investigation Unit of the Washington Hospital Center and maintained on a 300 Gm. carbohydrate diet throughout the whole period except when fasting was required. The patients with postgastrectomy hypoglycemia received their diet as 6 small meals spaced evenly throughout the day.

After an equilibration period on the diet for 3 days oral glucose (30 Gm. glucose per M^2 body surface) and intravenous glucose (0.5 Gm./Kg body weight) tolerance tests were performed on all patients. In those patients with functional hypoglycemia tolbutamide tolerance tests (1 Gm. sodium tolbutamide intravenously), and 72-hour fasts were also performed. In all these tests both serum glucose[7] and insulin[8] were measured.

During this period complete blood count, 24-hour urinary excretion of 17-ketosteroids and 17-hydrocorticoids, 24-hour urinary excretion of Na^+, K^+, Cl^-, liver function tests, and serum protein electrophoresis were measured by standard clinical methods in all patients.

Renal function tests (inulin, creatinine, PAH, uric acid and urea clearances) were also determined during 2 one-hour clearances by standard methods[9] on the 6 patients with functional hypoglycemia. Blood pressure and pulse rate were recorded 4 times a day, and weight daily.

Once these baseline measurements had been completed and the nature of the study and drug effects described to the patient (and necessary consent forms completed), the patients were started on diazoxide at a dose of 400 mg. per day in equal divided oral doses. The first 2 patients investigated received 800 to 1,200 mg. daily, but following severe symptoms the dose was reduced.

During diazoxide administration a 24-hour urine output was collected for estimation of Na^+, K^+, Cl^- and diazoxide output, and fasting blood sugar and diazoxide levels were measured[10] on alternate days. Fluid intake was maintained at approximately 2 liters per day.

Both before and after diazoxide administration 24-hour urinary excretion of catecholamines was estimated.[11] After 7 days of diazoxide therapy some of the general and special investigations were repeated, and this took a further 4 days. In 3 patients it was decided that diazoxide should be tried on a long-term basis, and these patients were seen in the outpatient department. One patient was readmitted for assessment of diazoxide sensitivity.

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Patients

In all, 10 patients received diazoxide, 4 patients suffering from postgastrectomy hypoglycemia and 6 patients with functional hypoglycemia.

Case 1. F. W. was 40 years old. In 1959, following a 11-year history of duodenal ulcer and intermittent gastrointestinal hemorrhage, a partial gastrectomy was performed. Two weeks following surgery attacks of severe epigastric fullness, weakness, sweating, and palpitations followed by hunger and occasional loss of consciousness began and have persisted ever since so that she could not work. In 1963 she was investigated by N.I.H. (Dr. J. Roth) with a diagnosis of postgastrectomy syndrome and severe psychoneurotic disorder. Oral glucose tolerance test revealed blood sugar level of 31 mg. at 1½ hours and normal intravenous glucose tolerance test.

Case 2. F. W. was 54 years old. In 1958 subtotal gastrectomy was performed following 16 years of abdominal pain and melena due to duodenal ulcer. Immediately following surgery she began to have attacks of nausea, palpitations, abdominal pain, and perspiration followed by diarrhea. Attacks were partly overcome by diet. Two-hour blood sugar level in an oral glucose tolerance test was 55 mg. with a normal intravenous glucose tolerance test.

Case 3. F. W. was 42 years old. Following many operations for abdominal pains including appendectomy, cholecystectomy, and many laparotomies, this patient had a pyloroplasty and vagotomy performed in 1962. Since this operation she has had attacks of cramplike abdominal pain, nausea, headache, and diarrhea. On admission she was taking trimethobenzamide for nausea and pethidine for abdominal pain. Xylose absorption was reduced at 3.9 Gm. in 5 hours (normal 6–11 Gm. in 5 hours). Intravenous glucose tolerance test revealed a classical high peak followed by the blood sugar falling to 50 mg. at 3 hours.

Case 4. W. M. was 64 years old. In 1962 a partial gastrectomy had been performed following a 2-year history of severe gastrointestinal bleeding. Since surgery he has complained of epigastric distention, weakness, trembling, and palpitations following meals. These symptoms disappear after 1 to 2 hours to be replaced by intense hunger, and they greatly interfere with his work as a furrier. Oral glucose tolerance test revealed a blood sugar level of 52 mg. at 2 hours. Intravenous glucose tolerance and xylose absorption tests were normal.

Case 5. N. M. was 39 years old. He had a 10-year history of dizzy attacks sometimes accompanied by palpitations and sweating lasting 30–60 minutes. They were not related to meals or exercise but more to anxiety. There has been no change in weight, and the patient is very active. On many occasions he was found to have a flat oral glucose tolerance curve, and a diagnosis of hyperinsulinism was suggested elsewhere. On admission an oral glucose tolerance curve was flat, the lowest level being 59 mg. 3 hours after the glucose with a marked increase in serum insulin levels. Intravenous glucose and tolbutamide tolerance tests and 72-hour fast were normal.

Case 6. W. M. was 18 years old. He was referred with an 8-month history of "blackouts" first occurring during exercise. He loses consciousness for 30–60 seconds, but there is no evidence of epilepsy. Sometimes he can overcome these attacks. These attacks came in groups and are worse when he is not eating regularly. His weight is steady. He occasionally takes dexamphetamine for scholastic studies and drinks quite heavily. A previous venereal infection was treated by penicillin. A flat glucose tolerance test was obtained on several occasions by us, but with a 3- to 4-fold increase in insulin secretion. Electroencephalograms remained normal even when the blood sugar was 41 mg. during the intravenous tolbutamide test. Intravenous glucose tolerance test and 72-hour fast were normal.

Case 7. W. F. was 21 years old. Following 2 attacks of infectious mononucleosis in 1965 she began to complain of severe asthenia, excessive sleepiness, tinnitus, headache, and lack of drive. Meals or exercise did not affect the symptoms, which became so severe that she dropped out of college. She has never lost consciousness, and her weight was steady. Clinical examination was normal, but a psychiatric interview carried out by Dr. J. Carranza of our Unit revealed that the symptoms were associated with environmental difficulties and suggestive of an underlying depressive reaction. Intravenous glucose tolerance and tolbutamide tests and 72-hour fasts were normal. Oral glucose tolerance test was normal except for a 4-hour blood sugar level of 66 mg., but lower values had been obtained elsewhere. An electroencephalogram tracing taken during the tolbutamide test when the blood sugar fell to 52 mg. was normal.

Case 8. W. F. was 50 years old. In 1939 this patient was diagnosed as being hypothyroid and since then has taken thyroid, at present 3–4 grains daily. Since 1945 she began to have attacks of nausea, headaches, and extreme fatigue diagnosed in 1949 as hypoglycemic when a flat glucose tolerance curve was discovered, and she started on a diet. In 1953 brucellosis was diagnosed. She improved on her diet, but the symptoms were severe enough to prevent her working. The weight is steady. Clinical examination was essentially normal. Routine electroencephalogram showed a basic alpha rhythm with short runs of low voltage 3–3½ c/s delta waves on the left side. Hyperventilation and photic stimulation evoked no additional abnormality. Thirty-five minutes after intravenous tolbutamide, when the blood sugar was 48 mg., a longer run of similar delta activity appeared in the left posterior temporal recording. Oral intravenous and tolbutamide tolerance tests and 72-hour fast were normal.

Case 9. W. F. was 43 years old. This patient has had Meniere's disease since 1959. In 1963 she suffered from fainting attacks associated with palpitations, perspiration, and weakness, mainly in the morning after waking. Symptoms are promptly relieved by taking liquid carbohydrate. There is no history of a convulsion. Oral glucose tolerance tests have all shown low levels 3–4 hours after glucose with symptoms. Past medical history included rheumatic fever and fibrocystic disease in both breasts. Intravenous glucose and tolbutamide tolerance tests and 72-hour fast were normal.

Case 10. N. F. was 16 years old. Since the age of 3 this girl has had grand mal epilepsy which at present occurs 3–5 times per year and for which she is taking sodium hydantoinate (Dilantin®). Her mother noticed that these attacks come on when she has missed a meal or following exercise or excitement. Clinical examination was normal. Oral and intravenous glucose and tolbutamide tolerance tests and 72-hour fast were normal. However, during the intravenous tolbutamide tolerance test when the blood sugar was 41 mg., a generalized slowing appeared in an initially normal electroencephalogram. This slowing then developed into a frequent bilateral synchronous discharge of high voltage 3–4 c/s regular waves in all leads. Thirty minutes later the recorder resumed normal activity when the blood sugar was 65 mg.

Results

In patients with postgastrectomy hypoglycemia (cases 1–4) classical oral glucose tolerance curves were obtained before diazoxide therapy. The high initial glucose levels at ½ and 1 hour were accompanied by a dramatic rise in insulin secretion and low blood sugar values 2–3 hours after glucose with a cut-off in insulin secretion. Accompanying the rise in glucose and insulin was a significant fall in serum K^+ values (figure 1). Ten days after diazoxide therapy the fasting blood sugar level was significantly elevated and there was no great fall in blood sugar levels at 2 and 3 hours. There was a marked reduction in insulin secretion, particularly within the first hour of the test, but no significant change in fasting insulin levels. However, the changes in serum K^+ levels were similar to the pretreatment test.

In patients with functional hypoglycemia (figure 2) 10 days of diazoxide treatment caused a marked change in glucose tolerance but no change in the fasting blood sugar levels. The insulin response was not as much changed as seen with the postgastrectomy patients. However, serum insulin levels were significantly reduced at 1 hour. Changes in serum K^+ levels were not altered.

Figures 3 and 4 show the intravenous glucose tolerance test before and after 9 days of diazoxide therapy in both groups. In the patients with functional hypoglycemia the rate of glucose disappearance between 30 and 60 minutes (as suggested by West and Wood[12]) is reduced from 2.68 ± 0.28 mg. per minute before diazoxide to 1.42 ± 0.33 mg. per minute after diazoxide ($P < 0.01$) with a marked reduction in insulin secretion within the first 15 minutes after the glucose infusion.

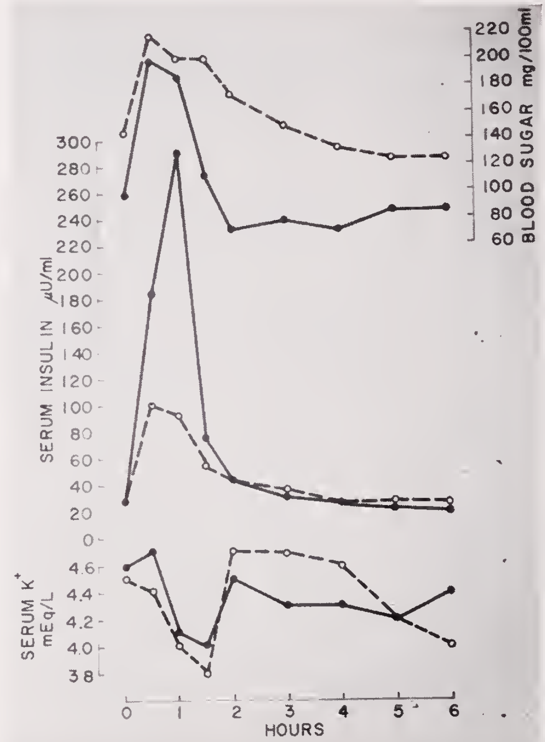

FIG. 1. Changes in blood sugar, serum insulin, and K^+ levels during 6-hour oral glucose tolerance tests in 4 patients with postsurgical hypoglycemia before (○---○) and after (●—●) 9 days of treatment with oral diazoxide.

In the 4 patients with postsurgical hypoglycemia there was no significant change in insulin secretion or glucose disappearance rate caused by diazoxide. However, these patients do not produce as much insulin in response to intravenous glucose as do the patients with functional hypoglycemia.

Tolbutamide tolerance tests were performed on the 6 patients with functional hypoglycemia before and after diazoxide, and the results in 5 are shown in figure 5. There is no significant change in blood sugar or insulin levels before and after diazoxide. However, in the sixth patient (case 6) a

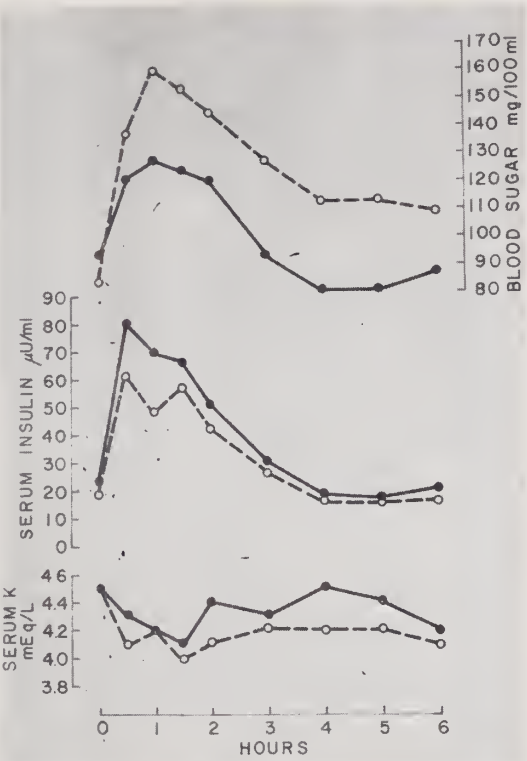

FIG. 2. Changes in blood sugar, serum insulin, and K^+ levels during 6-hour oral glucose tolerance tests in 6 patients with functional hypoglycemia before (○---○) and after (●—●) 9 days of treatment with oral diazoxide.

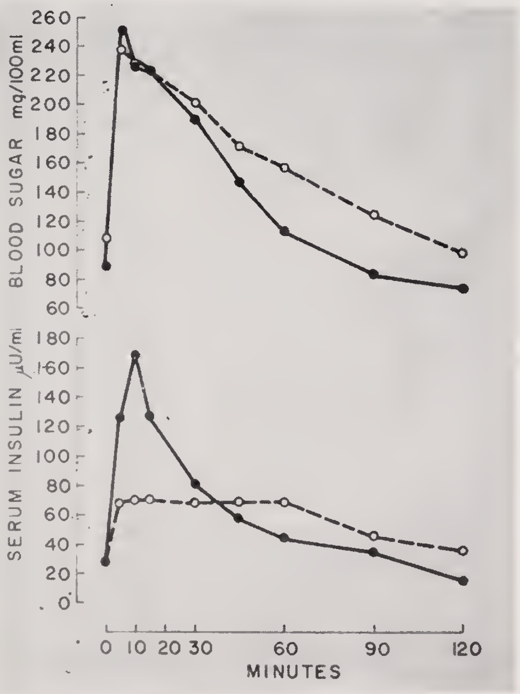

FIG. 3. Changes in blood sugar and serum insulin levels during intravenous glucose tolerance tests in 6 patients with functional hypoglycemia before (○---○) and after (●—●) 8 days of treatment with oral diazoxide.

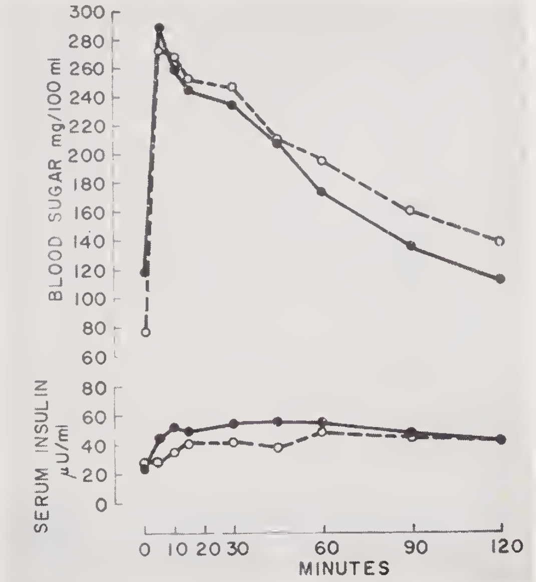

FIG. 4. Changes in blood sugar and serum insulin levels during intravenous glucose tolerance tests in 4 patients with postsurgical hypoglycemia before (○---○) and after (●—●) 8 days of treatment with oral diazoxide.

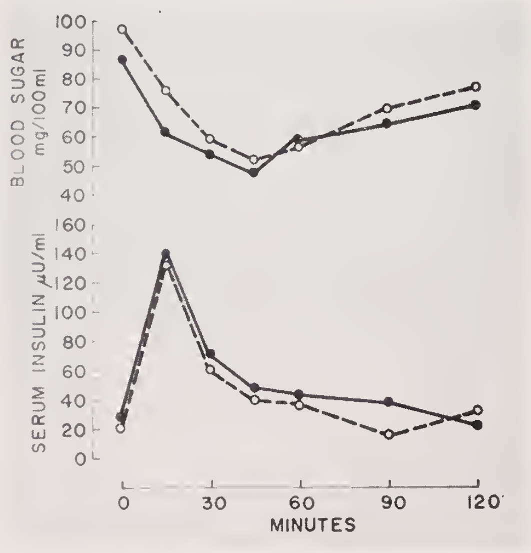

FIG. 5. Changes in blood sugar and serum insulin levels during intravenous tolbutamide tolerance tests in 5 patients with functional hypoglycemia before (○---○) and after (●—●) 10 days of treatment with oral diazoxide.

striking change did occur. Prior to diazoxide this patient had a normal tolbutamide tolerance test with the blood-sugar level falling to 41 mg. at 30 minutes and returning to within fasting range by 60 minutes with a 5-fold increase in serum insulin levels. However, after 10 days of diazoxide therapy he had a raised fasting blood sugar level (188 mg. and fasting serum insulin level 24 μU per ml.).

After intravenous tolbutamide the serum insulin levels became greater than 500 μU per ml. (top range of the assay) and remained at this level until 120 minutes after tolbutamide when the level had fallen to 292 μU per ml. This change was accompanied by a slight rise in blood-sugar levels to 210 mg. at 45 minutes, and then fell slowly to 147 mg. at 120 minutes. This is the only patient in whom we have observed this change, and his results have not been included in calculating the mean values shown in figure 5.

General changes in pulse rate, mean blood pressure, daily weight, 24-hour urine volume, and Na^+ K^+ Cl^- excretion are shown in figures 6 and 7 in all 10 patients. The pulse rate shown is the mean of 4 readings taken throughout the day, and the mean arterial blood pressure was calculated from the 8 a.m. reading with the patient recumbent for 5 minutes. On the average the patients gained 7 lbs. in weight. There was no change in blood pressure, but there was an increase in pulse rate. The most change in renal output occurred in the first 3 days with a fall in urine output accompanied by Na^+ retention and, to a lesser extent, K^+ retention but no change in Cl^- excretion. By the fourth and fifth days of diazoxide therapy these values had returned to pretreatment levels. In those patients developing clinical signs of edema this was usually observed by the third and fourth days.

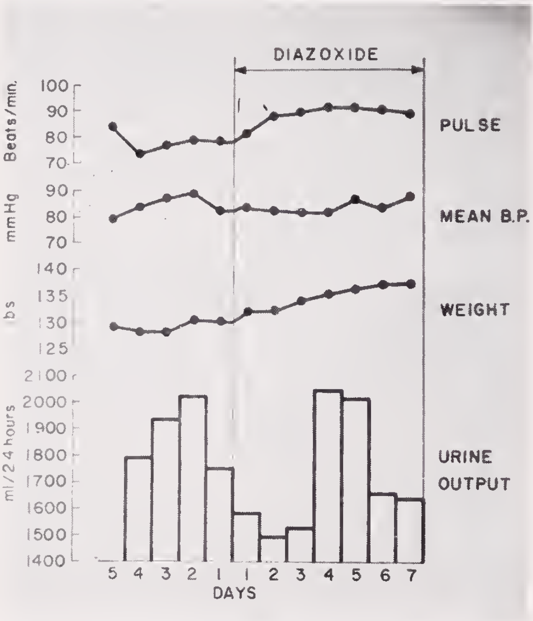

FIG. 6. Changes in pulse rate, mean blood pressure, weight, and urine output in all 10 patients.

Table 1 shows changes in the white blood cell count both before and during diazoxide therapy. One patient (case 1) developed granulocytopenia and a marked fall in platelet count with an erythematous rash and fever. She was later shown to be sensitive to diazoxide, and her case has been reported elsewhere.[13] Three other patients also showed some depression in the white blood cell count. There was no change in steroid excretion (17-ketosteroids and 17-hydroxycorticoids).

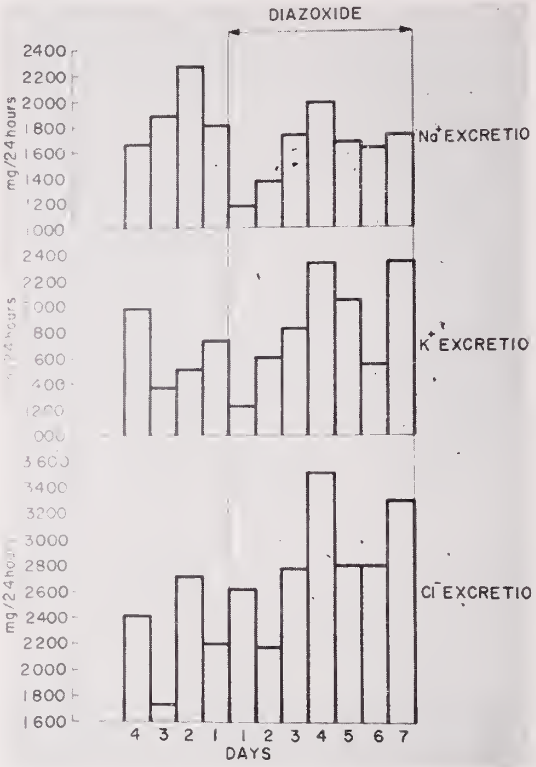

FIG. 7. Twenty-four-hour excretion of Na^+, K^+, Cl^- (before and during treatment with oral diazoxide in all 10 patients).

Changes in specific renal function tests are shown in table 2 performed before and after diazoxide therapy in the 6 patients with functional hypoglycemia. While there is a general reduction in all clearances measured, only in the PAH clearance is the change significant. Mean urine output during the clearances were 6.95 ml. per minute before and 6.32 ml. per minute after diazoxide administration.

Table 3 shows the changes in diazoxide levels in blood and urine during the initial days of therapy. Most of the dose administered is excreted by the kidney. However, not all is in the form of free diazoxide. From the second to third day of treatment there were 2 distinct forms in which diazoxide is excreted in the urine, a free and a conjugated form. Both are clearly shown by paper chromatography, but the measurements of diazoxide levels by UV-spectrophotometry cannot distinguish between them. Thus the urine figures represent total diazoxide excreted. By the end of the seventh day of treatment 90 per cent of the administered dose had been excreted in the urine.

There was no change in liver function tests in any patients receiving the drug, but 8 of the 10 patients showed a mild rise of 1 to 2 mg. in serum uric acid.

TABLE 1: Changes in White Blood Cell Count/cu. mm. Before and During Oral Diazoxide Therapy in 10 Patients

Case No.	Before	Day of Diazoxide Therapy						
		5	6	7	8	9	10	11
1*	6,201	—	—	—	3,274	3,403	2,920	—
2	4,830	—	—	—	—	—	3,368	3,116
3	6,361	4,765	—	4,783	—	—	—	3,750
4	6,608	—	—	6,517	—	—	—	—
5	9,036	—	—	8,477	—	—	—	—
6	6,508	6,834	—	5,570	—	—	—	—
7	4,824	—	—	—	4,992	—	—	—
8	5,698	—	—	4,391	3,877	3,417	—	4,734
9	8,926	—	—	7,359	—	—	7,287	—
10	3,261	—	5,045	—	—	—	4,586	—

* Reported elsewhere.

TABLE 2: Effect of Oral Diazoxide on Renal Function Tests in 6 Patients with Functional Hypoglycemia
(Mean values indicate mean of both first- and second-hour clearances in all patients.)

	Before Diazoxide			During Diazoxide			Sig. P
	1st hr.	2nd hr.	Mean	1st hr.	2nd hr.	Mean	
Inulin, ml./min.	98.5	105.3	101.8 ± 10.9*	79.0	83.3	81.2 ± 9.4	>0.1
Creatinine, ml./min.	97.7	110.5	104.1 ± 10.5	81.0	85.3	83.2 ± 10.0	>0.1
P.A.H., ml./min.	49.0	56.2	56.2 ± 48.2	36.1	38.3	37.2 ± 41.4	<0.05
Urea, ml./min.	55.6	68.4	61.9 ± 7.9	54.3	49.0	51.7 ± 6.3	>0.3
Uric acid, ml./min.	10.7	13.8	12.3 ± 2.0	8.8	8.4	8.6 ± 1.4	>0.1

* Mean ± Standard Error.

The excretion of catecholamines was measured as adrenalin and noradrenalin both before and during diazoxide treatment in 6 patients (patients 3 and 4 having postsurgical hypoglycemia and the others functional hypoglycemia). Diazoxide causes no great change in catecholamine excretion (table 4).

Despite changes in blood glucose and insulin due to diazoxide, symptomatic improvement was disappointing. Most patients complained of palpitations, weakness, headaches, nausea, anorexia, bad taste in the mouth, and feeling of bloatedness. All these symptoms were more marked in those 4 patients with clinical edema, although all but 1 patient increased in weight. Most of the symptoms cleared on stopping the drug. The patient who developed sensitivity to the drug developed pruritus, an erythematous rash, and fever.

Of the 6 patients with functional hypoglycemia diazoxide therapy was continued in only 1 patient (case 9) at a lower dose as an outpatient, and with intermittent diuretic therapy. This patient had a decreased incidence of attacks of sweating and dizziness in the morning, but after some weeks she noticed that the lumps in her breasts were enlarging and an increase of hair growth on the legs and arms. Both the latter signs regressed on stopping diazoxide medication. The audiogram in this patient's Meniere's disease was not affected. Case 10 had a grand mal epileptic seizure on diazoxide therapy but had discontinued her sodium hydantoinate during her admission.

Two of the 4 patients with postsurgical hypoglycemia said they felt better (cases 2 and 3) with fewer severe attacks and much less diarrhea. Both patients were continued on lower doses of diazoxide and diuretics, but in both the water retention and palpitations became too persistent and the drugs were discontinued. One patient noticed an increase of hair growth on the hands and face.

TABLE 3: Levels of Diazoxide in Blood (mg. per 100 ml.) and Urine (mg. per 24 hours) During Treatment

Case No.	Wt. lbs.	Dose mg./day	Drug Level	Days of Diazoxide Treatment									
				1	2	3	4	5	6	7	8	9	10
1	103	800–1,200	blood	—	—	—	7.8	—	10.2	—	—	13.6	13.0
			urine	440	580	660	560	—	730	630		—	—
2	100	800	blood	—	9.4	—	—	—	16.0	—	15.5	—	—
			urine	170	290	370	420	530	620	610	—	—	—
3	126	400	blood	—	2.5	—	4.5	—	1.6	—	2.2	2.4	—
			urine	244	321	391	487	317	430	292	—	—	—
4	146	400	blood	—	1.6	—	1.7	—	2.7	—	5.0	4.9	—
			urine	400	550	461	392	—	645	—	—	—	—
5	169	400	blood	—	1.8	—	1.2	—	1.5	—	1.5	1.9	1.8
			urine	220	481	455	324	241	428	284	—	—	—
6	160	400	blood	—	1.2	—	1.5	—	4.0	—	3.0	3.5	3.0
			urine	357	—	401	557	468	320	417	—	—	—
7	99½	400	blood	—	10.5	—	11.8	—	11.4	—	8.0	11.8	7.3
			urine	160	178	303	385	481	627	551	—	—	—
8	134	400	blood	—	5.1	—	5.1	—	4.9	—	3.8	9.4	4.7
			urine	358	417	459	538	609	573	456	—	—	—
9	131	400	blood	—	3.8	—	4.0	—	4.0	—	4.3	4.5	3.5
			urine	279	329	—	475	384	—	376	—	—	
10	107	400	blood	—	2.6	—	2.3	—	3.5	—	4.1	5.2	6.6
			urine	—	—	185	281	282	197	—	—	—	—

TABLE 4: 24-hour Urinary Excretion of Adrenalin (Adr) and Noradrenalin (Noradr) in mg./24 hours Before and During Diazoxide Therapy in 6 Patients

Case No.	Catecholamines	Before	24-hour Urinary Catecholamine Excretion mg./24 Hours Diazoxide Treatment						
			1	2	3	4	5	6	7
4	Noradr	21.1	13.5	14.1	28.4	13.6	19.6	19.2	—
	Adr	2.5	2.9	1.5	1.2	1.1	3.0	2.6	—
5	Noradr	44.0	39.5	89.5	59.4	138.6	—	64.2	25.2
	Adr	2.6	3.4	5.1	2.4	10.8	—	4.3	21
7	Noradr	25.3	25.4	22.5	7.0	35.4	44.4	49.4	20.1
	Adr	1.2	0.2	0.8	1.0	10.0	0.6	3.2	4.7
8	Noradr	56.2	89.0	43.7	49.6	46.8	54.4	71.0	55.1
	Adr	5.2	4.5	7.3	8.6	8.2	7.9	3.1	7.5
9	Noradr	34.0	66.2	—	—	—	—	86.1	33.4
	Adr	0.4	3.7	—	—	—	—	1.4	1.3
3	Noradr	48.9	56.8	36.9	64.4	20.8	54.5	12.6	38.5
	Adr	5.8	3.3	3.0	4.4	1.8	4.1	1.0	1.2

Discussion

This study was in no way blind. Before beginning our studies with diazoxide the effects and likely side-effects of the drug had to be fully explained to the patient. This would perhaps explain the high incidence of subjective symptoms in the patients. Most of the patients we studied had been complaining of their symptoms for many years and had become psychologically dependent on them. Of the 2 groups of patients we studied, those patients with postsurgical hypoglycemia seemed helped to the greatest degree. Overall symptomatic improvement in the patients was disappointing.

One method of reducing the incidence of symptoms due to diazoxide would be to combine the drug with a thiazide diuretic at the onset of weight gain.

The biochemical effects of diazoxide are clearly shown. Hyperglycemia is accompanied by lower serum insulin levels best demonstrated in the oral glucose tolerance tests in postsurgical hypoglycemic patients and in the intravenous glucose tolerance tests in the functional hypoglycemic patients. The K^+ shift from the extracellular flood into the cell which is most marked in the glucose tolerance test in the postsurgical patients does not seem to be dependent on the rapid changes in glucose and insulin levels, since the same changes in serum K^+ levels occur after the glucose and insulin levels have been greatly changed by diazoxide.

Our results also rule out increased secretion of catecholamines, steroids, and impaired liver function as causes of diazoxide-induced hyperglycemia. However, the results do not rule out that the hyperglycemia could be due to an increased sensitivity to normal levels of catecholamine secretion. Rennick et al[14] have shown that diazoxide and the diuretic benzothiadiazines greatly reduce the tubular excretion of catecholamines in the chicken, but our work does not support these findings in man.

The renal effects of diazoxide are more marked in the first 3 days of therapy. During this time the blood level of the drug is usually rising. Urinary excretion of diazoxide is also increasing and during these initial days is comprised mainly of the "free" form. After 3 to 4 days a greater amount of the conjugated form of the drug is being formed, probably by enzyme induction. One may speculate that the renal effects are due to the "free" form and are therefore maximal at the beginning of therapy, while the hyperglycemic effects of the drug are independent of conjugation. This may also explain why the decreases in the specific renal function tests were not significant on the eighth day of diazoxide therapy. The significant fall in PAH clearance may not fully indicate a fall in effective renal plasma flow, and some authors have suggested that PAH and diazoxide are excreted by the same tubular mechanism and may compete for excretion at this site.[15]

The anomalous result of the intravenous tolbutamide tolerance test in patient 6 might be explained by postulating that the insulin released, although biologically inactive, was able to cross-react in the immunologic estimation of insulin. There was no significant fall in blood pressure during diazoxide therapy, and this is in marked contrast to the effect of the drug when administered intravenously.[16] However, the increase in pulse rate observed may indicate peripheral vasodilatation in some patients.

Some patients with functional hypoglycemia did not improve symptomatically, although there was increase in blood sugar levels. This might be because the untoward symptoms of diazoxide administration replaced the hypoglycemic symptoms.

Most other investigators administering diazoxide orally also encountered a high incidence of side-effects. The largest trial during the initial use of diazoxide as an antihypertensive agent in 30 patients lasting from 8 to 50 weeks reported a weight gain in 26 patients, gastrointestinal symptoms (from anorexia to nausea and vomiting) in 15 patients, lacrimation in 6 patients, and transient cardiac arrhythmia in 4 patients.[17] One patient with an islet-cell carcinoma on 300 mg. diazoxide per day had persistent nausea and vomiting.[18] Samols and Marks[2] treated 4 patients with hypoglycemia with diazoxide. The 1 adult had dependent edema and hair growth, while the children complained of anorexia, nausea and vomiting, mild sodium retention and edema, and excessive hair growth. Hutcheon and Barthalmus[19] using diazoxide as an antihypertensive agent at a dose of 300 mg. per day had variable changes in K^+ excretion but retention of Na^+ and water and 2 cases of paroxysmal supraventricular tachycardia. One patient with von Gierke's disease treated with diazoxide with 100–200 mg. per day developed a petechial eruption, facial erythema, and generalized pruritus which cleared on stopping diazoxide but recurred with attempts to reintroduce the drug.[20] However, on short-term investigatory studies Graber et al[21] reported no side-effects.

Case 1 demonstrates one of the more serious side-effects of diazoxide, and we advise careful supervision of hematologic parameters.

Although diazoxide does not seem to be indicated in the treatment of functional hypoglycemia, the drug may be of some use in patients with postsurgical hypoglycemia if administered with a diuretic to overcome water retention.

Summary

Diazoxide, a hyperglycemic, nondiuretic benzothiadiazine, has been used in the treatment of hypoglycemia secondary to gastric surgery and functional hypoglycemia. Observations were made on blood glucose and insulin, glucose and tolbutamide tolerance, renal function tests, and catecholamine and steroid excretion in the urine.

While patients with the postgastrectomy syndrome showed some symptomatic improvement, patients with functional hypoglycemia did not. Both groups had severe side-effects, both subjectively and by water retention, most marked in the initial 3 days of treatment. In 1 patient severe granulocytopenia occurred.

Both groups showed marked changes in glucose tolerance and some degree of inhibition of insulin secretion. These changes showed no correlation with increased catecholamine or steroid excretion or impaired liver function.

Diazoxide is excreted largely by the kidneys. By the third and fourth days this is mainly as a conjugated form, when the renal effects of the drug are less.

The future use of diazoxide in these types of patients has been discussed.

These studies would not have been possible without the help of our nurses, Miss Johanna Carey, Miss Janet Koch, Miss Erica Rudin, Mrs. Hildegarde Jorgensen, and Miss Joan Roche, and our technicians led by Miss Alison Grant, Mr. A. Baukal, and Mrs. Kathleen Weinhardt.

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Paradoxical Embolism in Congenital Heart Disease

Report of Two Cases

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PARADOXIC OR CROSSED EMBOLISM is defined as a condition in which emboli derived from the systemic venous system reach the systemic arterial system by passing through an abnormal communication between chambers of the heart.[1] It was first described in 1860 by Cohn, and again in more detail by Cohnheim in 1877. Zahn first demonstrated crossed embolization at autopsy in 1882.[2] This form of arterial embolism is only briefly mentioned in most textbooks, and only sparsely reported in the literature. Gill and Dammin[3] found only 101 cases in the world literature by 1958. It undoubtedly occurs more commonly than described and is a frequently missed diagnosis even though its occurrence is limited to special anatomic and physiologic circumstances and presents a rather consistent clinical picture.

The following 2 patients represent examples of congenital heart disease complicated by paradoxical embolism.

Case 1. E. G., a 17-year-old boy, was admitted in July of 1962 after an acute occlusion of the left femoral artery. He had been well until 6 months prior to admission when he complained of transient episodes of numbness and weakness in his extremities. Two weeks prior to admission he sustained a blow to the right leg without visible injury. Three days prior to admission he had a brief episode of unconsciousness, following which he was aphasic with paresis of the right side of his body. In the ambulance en route to the hospital he developed a severe pain in the left leg and had a grand mal seizure.

He was described as cyanotic as an infant and had been mentally retarded since birth. There were no symptoms related to his cardiopulmonary system.

He was tall, thin, and of sallow complexion. There was minimal cyanosis of the lips and nailbeds and clubbing of the digits in all extremities. The chest was enlarged in the anteroposterior diameter with a bulging of the lower sternum and left chest-cage. There was a palpable right ventricular lift of the lower sternum and a systolic thrill at the apex. The second sound over the pulmonic area was accentuated and single. There was a short, harsh systolic murmur, grade II/VI, over the base of the heart. A grade V/VI holosystolic murmur radiated from the lower sternum to the apex, left axilla and interscapular area. A sinus tachycardia was present. The left leg was cold, cyanotic, and pulseless. The boy was aphasic and had a right-sided hemiparesis.

An electrocardiogram showed right ventricular hypertrophy and left axis deviation. A chest X-ray film and fluoroscopy revealed right ventricular enlargement and increased pulmonary vascularity.

An embolectomy was performed on admission, and a long clot was removed from the femoral artery, which was sterile on repeated cultures. The patient was discharged on anticoagulants and referred to the National Heart Institute, where definitive studies were performed:

Cardiac catheterization:
Right atrium = 4 mm. Hg
Right ventricle = 20/4 mm. Hg
Pulmonary artery = 20/10 mm. Hg
Left atrium = 4 mm. Hg
Left ventricle = 100/6 mm. Hg
Left brachial artery = 100/70 mm. Hg

A dye curve revealed a left-to-right and a right-to-left shunt at the atrial level. A left ventricular angiogram revealed mitral regurgitation and an enlarged left atrium, with a left-to-right shunt at the atrial level and a localized area of muscle hypertrophy anterior to the mitral valve. An atrioventricular canal defect seemed the most likely diagnosis, and, on March 20, 1963 the patient underwent corrective surgery on cardiopulmonary bypass. At surgery a common atrium and cleft mitral valve were found and successfully repaired.

Case 2. P. S., a 21-year-old white woman, was admitted because of cyanosis and vaginal bleeding. She was noted to be cyanotic shortly after birth and had had 3 episodes of pneumonia as a child. She noted fatigue and squatted to relieve dyspnea. Although she was small and thin for her age, she was larger than her healthy siblings. She had always been active physically until the year of admission when she noted progressive fatigue and dyspnea on exertion.

There was marked clubbing and cyanosis of all extremities, with the blood pressure 90/75 in the arms and 110/80 in the legs. The neck veins were flat. There was a prominent right ventricular lift. The apex beat was localized in the fifth left interspace at the mid-clavicular line. There was no thrill. The second sound over the pulmonic area was accentuated but not split. A loud ejection sound was heard over the lower sternal border. A continuous bruit was heard over the right infraclavicular area. The hematocrit was 54.5 per cent. An electrocardiogram showed right ventricular hypertrophy. X-ray and fluoroscopy revealed right ventricular enlargement and a small right-sided aorta and diminished pulmonary vascularity.

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The patient gradually developed orthopnea which responded to digitalization and phlebotomy. In September of 1963 she had an incomplete abortion. In October of 1963 she had 2 episodes of pleuritic chest pain with fever and a rub diagnosed as pulmonary infarction. Following this there were several episodes of light-headedness and clonic movements of the left upper extremity with weakness. Two grand mal seizures occurred. Neurologic examinations, including cerebrospinal fluid and brain scan, were normal; however, the electroencephalogram was abnormal, with slowing and sharp activity over the right posterotemporal and occipital areas. Angiography showed the pulmonary artery to arise from a common trunk with small branches.

Cardiac catheterization: The catheter moved from the right atrium to the right ventricle to the aorta:

Right atria = 0 mm. Hg
Right ventricle = 110/10 mm. Hg
Aortic root = 110/55 mm. Hg
Brachial artery = 110/55 mm. Hg

A dye dilution curve following injection into the right ventricle and superior vena cava showed a right-to-left shunt and a left-to-right shunt, with sampling from the aortic root. There was a step-up in oxygen saturation from the right atrium into the right ventricular outflow tract.

At thoracotomy the patient was found to have a type IV truncus arteriosus or pseudotruncus with an atretic pulmonary artery and a small atrial and ventricular septal defect. Pulmonary blood flow originated from large bronchial arteries. The left subclavian artery was anastomosed to the pulmonic artery, but 12 hours postoperatively the anastomosis opened and exsanguination occurred.

Discussion

The first patient represents a well-defined example of venous thrombosis in the right saphenous system, with embolization into the systemic circulation through a large atrial defect. There was occlusion of both the left femoral and left middle cerebral arteries. The second patient also had embolization to the pulmonary and cerebral circulation associated with a right-to-left shunt. The possibility of thrombosis in situ secondary to polycythemia is not definitely excluded.

The majority of instances of paradoxical systemic embolism are preceded by episodes of pulmonary embolization. Indeed, a clinical situation in which there is venous thrombosis followed by pulmonary embolization, recovery, and then infarction of another organ, particularly the brain, is characteristic of paradoxical embolization. Elevation of right-sided intracardiac pressure usually secondary to pulmonary embolization is basic to the pathophysiology of this disorder.

Under normal circumstances the pressure in the left atrium is equal to or slightly exceeds that of the right atrium and maintains a flaplike valve over the foramen ovale. Pulmonary embolization, especially when associated with cardiac decompensation, is accompanied by an elevated right-sided intracardiac pressure with a reversal of flow of blood and opening of the defect. In the presence of massive embolization the left atrial pressure falls, still further enhancing crossed embolization.[4]

Haggart and Walker[5] demonstrated by stepwise occlusion of pulmonary artery flow that they could increase pulmonary and right-sided intracardiac pressure and eventually decrease systemic pressure. An occlusion of 60 per cent of the pulmonary circulation was needed to cause a sufficient rise in the right atrial pressure to establish a right-to-left shunt.

Elliott and Beamish[6] point out that opening of the patent foramen is palliative and acts as a safety valve. When a large clot occludes the foramen ovale there is sudden death.

Emboli arising as a venous cast are usually large enough to occlude a defect of 0.7 cm. In most routine autopsies the foramen ovale is closed, but in one series at least 39 per cent of specimens had an opening large enough to permit the passage of a probe (0.2 cm.), and 6 per cent had a defect large enough to permit passage of a lead pencil 0.7 cm. in diameter.[4]

Thirty-five per cent of Gill's autopsied cases had a large embolus occluding the foramen ovale. Zahn's original case report described this very situation.[3]

Most of the reported cases have occurred in middle-aged women, especially postoperatively, and usually had multiple visceral involvement, most often the lungs, brain and kidneys.

Gill and Dammin[3] reviewed 101 cases of paradoxical embolism, of which only 3 patients survived. Out of the 101 cases 97 per cent had patent foramen ovale, and the remainder an intraventricular septal defect. Abbott et al[7] analyzed 1,000 cases of congenital heart disease at necropsy and found 12 cases of paradoxical embolism associated with patent foramen ovale, intraventricular septal defects, and patent ductus arteriosus.

Crossed embolization is reported to be more common with an interventricular septal defect, especially when associated with a right-sided aorta, than with an intra-atrial septal defect.[8]

Thirty-five per cent of the cases in one series had no pulmonary embolization at autopsy. The necessary pressure changes in some patients were the result of chronic lung diseases such as tuberculosis, sarcoidosis, and emphysema. However, in the majority of instances there was no obvious cause, and it is speculated that very small emboli associated with reflex changes or vasoconstrictor humoral agents, such as serotonin, have been present, causing a more marked rise in pulmonary than systemic pressure.[3]

Thompson and Evans[4] pointed out that embolic material is not always thrombotic in origin. They presented an interesting example of a solid tumor embolus arising as a malignant teratoma of the testicle, entering the venous system, crossing into the systemic circulation via a patent foramen ovale, and terminating by acutely occluding a coronary artery. They classified paradoxical emboli as arising from thrombosis, tumor, or septic material. Thrombosis was by far the most common and usually involved the deep veins of the leg and pelvis. Tumor emboli, on the other hand, were extremely rare, and most reported cases are associated with metastatic lesions of the lung, suggesting that the tumor may have traversed the pulmonary circulation.[6] The lung and thyroid were the usual sources of tumor emboli.

Emboli of the systemic circulation have many possible sources. They may be dislodged from thrombi in the left side of the heart associated with disease of the aortic or mitral valve, or mural thrombi overlying a myocardial infarction. They may also arise from fragments of atheromatous ulcers or from a region of aneurysmal dilatation of the aorta or other large artery. The pulmonary veins occasionally transport emboli from a diseased lung to the left side of the heart, especially from abscesses, pneumonia, or tumors.[9]

In a series of 134 peripheral arterial emboli, only 2 were paradoxical, whereas 52 were associated with rheumatic heart disease and 55 with arteriosclerotic heart disease. However, this series excludes cerebral vascular embolization, which was cited by several authors as the most common peripheral site of paradoxical embolization.[10]

Renal artery embolization and infarction as reported by Gill and Dammin is frequently asymptomatic. A frequent complication of this disorder is brain abscess. Robbins[11] reported 3 cases of paradoxical emboli and brain abscess in a "tetralogy" and noted that either a sterile or infected embolus may cause an abscess. In the former, a plaque of encephalomalacia is formed which becomes infected during bacteremia.

Summary

Two cases of multiple embolization in patients with congenital heart disease have been presented as examples of paradoxical embolization. Pertinent anatomic and pathophysiologic data have been described.

Ninety-seven per cent of cases occurred in the presence of patent foramen ovale. The embolic material most commonly originated from pelvic and leg veins. The lungs, brain, and kidney are common sites of concurrent embolization.

Pulmonary hypertension secondary to embolization produced a right-to-left shunt through an opened foramen ovale.

Venous thrombosis complicated by pulmonary embolization and later by peripheral arterial embolization, especially cerebral, is pathognomonic of paradoxical embolization.

All hemodynamic studies and cardiac surgery were performed by the Section of Cardiology, National Heart Institute, Bethesda, Maryland.

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Intracranial Extradural and Subdural Empyemas

Report of a Case and Review of the Literature

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THE OCCURRENCE of extradural and subdural collections of pus has been known since 1773, when Richter[1] reported the case of a 50-year-old man with chronic frontal sinusitis who developed hemiparesis and aphasia. Exploration of the sinus revealed a perforation of its posterial wall with pus behind it, possibly lying in the subdural space. During the past 25 years there have been many significant changes in the frequency, etiology and prognosis of these cases. The present case is being reported because it illustrates the possibility of iatrogenic influences and the occurrence of large extradural and subdural collections of pus. It affords an opportunity to review briefly present-day management.

Report of Case

The patient, a 16-year-old boy, was in good health until 3½ weeks before admission, when he began to experience a constant bifrontal headache, which later radiated back over the vertex to the occiput. He also complained of nasal congestion and generalized malaise. After 7 days the pain in his head was so severe that he was taken to the emergency room of another hospital, where he was found to be febrile, and was given nose drops plus tetracycline, of which he took 2 capsules every 4 hours for 12 days. He remained febrile, sweated at night, and lost 5 lbs. On January 18, 1968 he saw his own physician for the first time and was immediately admitted to the Washington Hospital Center.

He was an awake, fully-oriented, sick boy lying in bed with a drawn expression on his face and closed eyes; he screwed up his forehead in pain. The forehead and vertex were tender to palpation and there was a slight degree of neck stiffness. The temperature was 102.4°F, the pulse rate 100, respiratory rate 20, and the blood pressure 100/70. He could distinguish smells normally with the left nostril but made errors with the right. The pupils were moderately dilated, equal in size, and reacted briskly to light and accommodation. The optic disks were clearly outlined, but the veins were full and did not pulsate. The rest of the cranial nerves were normal. The patient allowed his outstretched upper limbs to drift downward, but power was good in all muscle groups. Deep tendon reflexes were normally brisk and equal on both sides. Plantar reflexes were flexor bilaterally. When he stood there was marked swaying without predilection for either side; on these occasions he complained of severe exacerbation of his head pain, which became throbbing, and he crumpled at the knees. Sensory examination was not remarkable.

Initial laboratory data: hemoglobin 11.5 Gm., hematocrit 34 per cent, sedimentation rate 28 mm. (corrected), white blood cells 22,290 with 93 per cent polymorphonuclears, 4 per cent lymphocytes, and 3 per cent monocytes, sickle-cell preparation negative, urinalysis normal. Spinal puncture produced clear, colorless fluid with an opening pressure of 290 mm., protein 58 mg., 5 lymphocytes per cu. mm., sugar 83 mg. with a simultaneous blood sugar of 117 mg. A blood culture on January 19, 1968 was negative. PPD was negative. X-ray films of the skull showed no abnormalities of the paranasal sinuses, cranial vault, or sella turcica; the pineal body was not identified. Roentgenograms of the chest were normal.

On January 19 the patient was delirious. The optic disks were now unclear and the veins more engorged, especially on the left. Neck stiffness was still present. Brudzinski's sign was present, but Kernig's sign was not elicitable. An electroencephalogram contained a background rhythm of 7 cycles per second with some intermixture of slower theta activity. It was significantly flatter in amplitude on the left, and many 1.5–2 cycles per second high voltage delta waves appeared at random and in runs in all leads on the left. A technetium brain scan showed marked increase of radioactivity along the convexity of the left hemisphere (figure 1).

The history and physical findings were considered typical of an extra- or subdural empyema. Left carotid arteriography in the lateral projection revealed an avascular radiolucent space over the frontal portion of the hemisphere, with displacement downward of the superior sagittal sinus, indicating an extradural collection of fluid (figures 2-A and 2-B). In the anteroposterior projection the anterior cerebral artery was shifted minimally to the right (figure 3). Immediately

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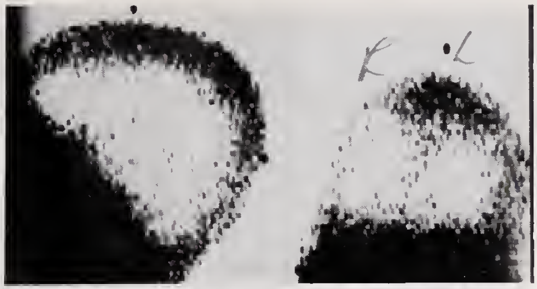

FIG. 1. Technetium brain scan in lateral and PA projections. Increased radioactivity along the convexity of the left hemisphere.

following the procedure burr-holes were made about 2.5 cm. to the left of the midline, 3 cm. anterior, and 3 cm. posterior to the coronal suture. As soon as the burr penetrated the inner table of the skull, thick yellow pus exuded freely from an extradural space. Approximately 35 ml. of pus was obtained. Gram's stain showed chains of gram-positive cocci, which were shown on culture to be anaerobic streptococci sensitive to all the common antibiotics. The patient had been started on 1,000,000 units of penicillin every 2 hours and on January 20 this was changed to sodium cephalothin (Keflin®), 1 Gm. every 4 hours.

On January 20 and 21 the patient was still febrile, with temperatures above 101°F, white blood cells 19,300 with 94 per cent polymorphonuclears and 6 per cent lymphocytes. On January 22 the temperature was 102°F. The patient reacted only to painful stimuli. The left pupil was dilated and failed to react to light. Seizures occurred, involving the right side of the face and neck. The spinal fluid contained 44 cells of which 26 were polymorphonuclears and 18 lymphocytes. The arteriograms were reviewed, and because of a suspicion of extradural pus on the right side, a right parasagittal burr-hole was made, but there was no extradural pus. The previous burr-holes were explored on the left, and an additional temporal opening was also made. Again, no extradural pus was encountered. The dura was not opened for fear of spreading the infection intracranially.

Postoperatively the patient was given penicillin, 1,000,000 units every 4 hours, sodium cephalothin, 1 Gm. every 4 hours, and dexamethasone sodium phos-

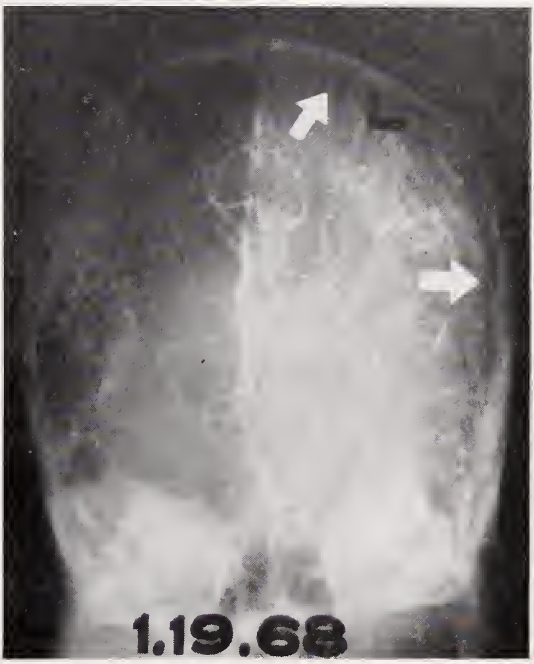

FIG. 3. January 19, 1968. Left carotid angiogram AP projection. Crescentic avascular space over left hemisphere extending from vertex to temporal region; only minimal shift of anterior cerebral artery.

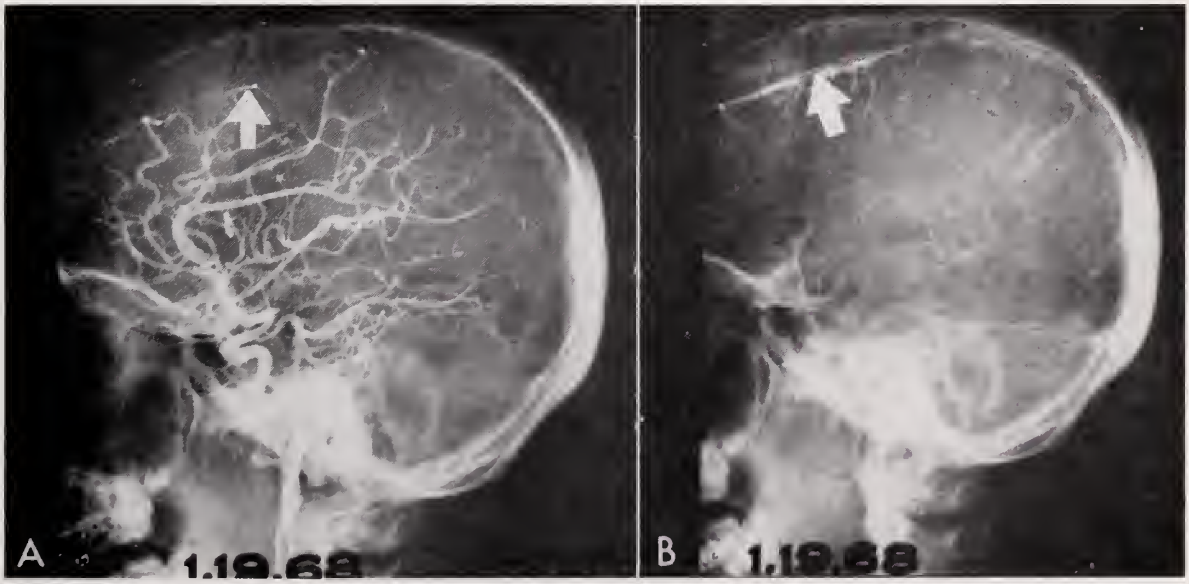

FIG. 2. January 19, 1968. Left carotid angiogram in lateral projection: A, arterial phase; B, venous phase. Cortical arteries and superior sagittal sinus are displaced downward by epidural empyema.

phate (Decadron®), 2 mg. every 4 hours. On January 23 the left pupil remained larger than the right, but both reacted to light. There was bilateral papilledema, left greater than right, and a hemorrhage was seen on the right disk. All his extremities could be moved, and the tongue was protruded on command. The neck was less rigid. On January 24 the patient was more alert and coöperative and obeyed simple commands. The temperature was normal. Spinal tap on January 30 revealed clear, colorless fluid with an opening pressure of 260 mm. It contained 24 mg. of protein, 28 lymphocytes per cu. mm., and 80 mg. of sugar. The blood sugar was 100 mg. On January 31 sodium cephalothin was discontinued. Later that day the patient became restless and complained of severe headaches; the temperature was 98.6°F. A second left carotid arteriogram showed a new avascular area suggestive of a subdural empyema (figures 4-A and B and 5).

A left temporoparietal craniectomy was performed. The dura underlying the previous temporal burr-hole was seen to be quite tense. It was coagulated in the center and opened. A dark membrane was revealed, which was opened, and a specimen was removed for examination. Thick yellowish white pus exuded in tremendous amount. No bacteria were found with Gram stain, and none grew at 24 or 48 hours, aerobically or anaerobically. The burr-hole was enlarged to a 3 cm. diameter craniectomy in the temporoparietal area. The brain was noted to be separated by a cavity at least 2 cm. deep. The pus was washed out with copious amounts of saline and with bacitracin solution (50,000 units to 50 ml. at first, and then 50,000 units to 250 ml.). The brain was observed to begin to pulsate and before the end of the procedure had risen to the surface of the dura. Penrose drains were inserted, 1 in the superior and 1 in the temporal area, and a no. 8 French catheter was placed in the anterior part of the cavity. The patient recovered well from the operation but was noted to have a degree of expressive dysphasia. His pupils were equal and reacted to light, and he was afebrile. The dosage of dexamethasone was increased to 4 mg. every 4 hours, and the antibiotics were maintained in their previous dosage.

On February 2 the temperature was 100°F. The patient carried out simple commands promptly and denied headaches. There was no weakness in any extremity. The pupils were equal and reacted briskly to

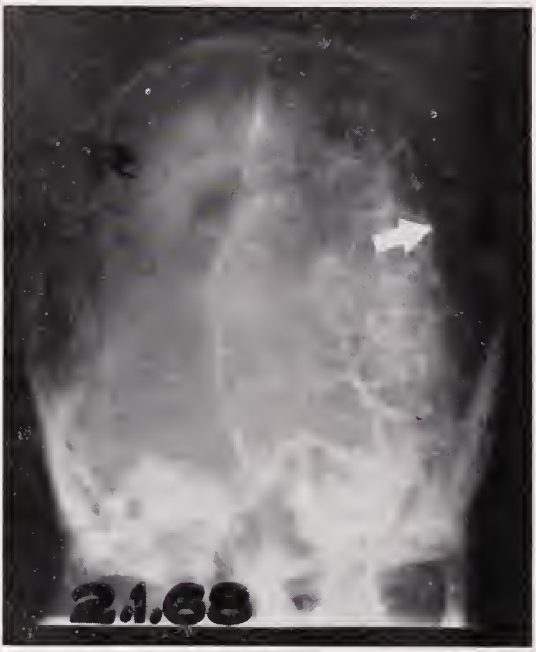

FIG. 5. February 1, 1968. Left carotid angiogram, AP projection. Same study as in figure 4. Large subdural empyema over left convexity extending into temporal fossa; marked shift of anterior cerebral artery.

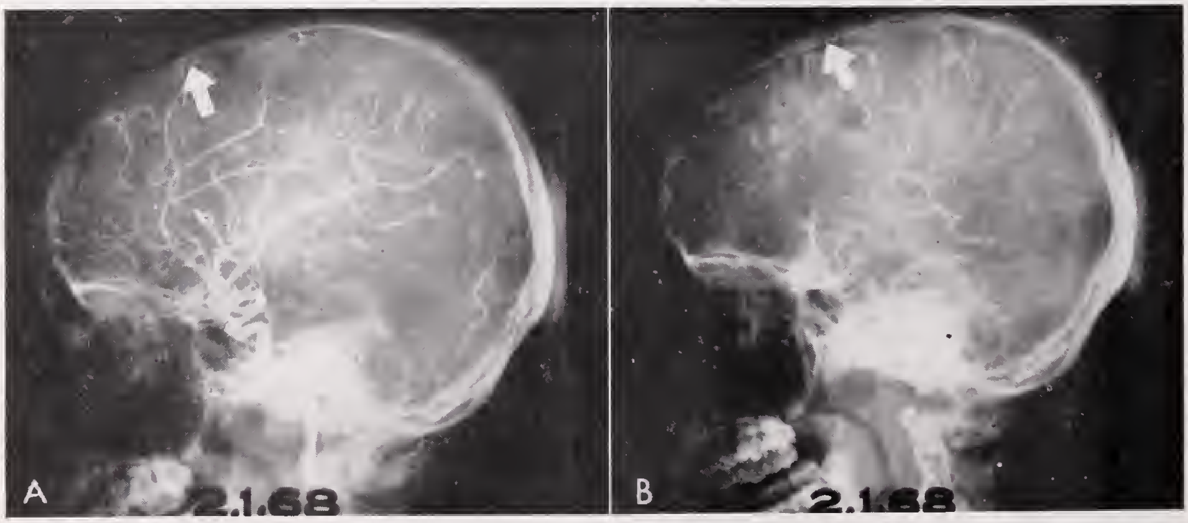

FIG. 4. February 1, 1968. Left carotid angiogram in lateral projection: A, arterial phase; B, venous phase. No abnormalities. Cortical arterial branches extend to inner table, and superior sagittal sinus is not displaced. Several burr-holes are visible.

light. The wound was irrigated again with bacitracin solution. On February 4 the condition had improved greatly and the remaining tubes were removed. On February 6 antibiotics were discontinued and the dexamethasone was terminated by steps. Following this the patient made an uneventful recovery. He was discharged asymptomatic on February 17, has remained well to date, and has resumed attendance at school.

Anatomic Considerations

The dura mater consists of 2 layers of tough fibrous membrane separated by a layer of vascular tissue. The fibers of the 2 layers run obliquely to each other. The outer, or endosteal, layer is adherent to the inner table of the calvarium and becomes continuous with the pericranium at the various foramina and through the sutures. It sends fibrous processes and blood vessels into the bone, and because of these the dura presents a shaggy appearance when stripped from it. The dura becomes more adherent with increasing age. The inner, or meningeal, layer sends folds inward to form the falx cerebri, tentorium cerebelli, diaphragma sellae, and the roof of the cavum trigeminale. It is separated from the outer layer of arachnoid epithelium (del Rio Hortega cells) by a potential space, the subdural space, which normally contains at most a slight film of serous fluid but is capable of becoming distended by blood, pus, or other fluid.

Extradural pus can extend backward from the frontal sinus, stripping the dura away from the bone. It may be unilateral, but pus may also pass to the opposite side, depressing the superior sagittal sinus as it does so. There is a tendency for extradural pus secondary to otitic infection to collect over the tegmen tympani; direct extension into the brain substance or destruction of meninges with localized empyema are also apt to occur in this situation.

The degree of distensibility of the subdural space varies in its different parts. It is tethered to the superior sagittal sinus superiorly and to other sinuses inferiorly by the pacchionian granulations and to the base of the skull by the passage across it of cranial nerves, blood vessels, and the pituitary stalk. These anatomic features and the particular primary site of infection, which is usually the frontal sinus, influence the region in which pus tends to collect. The site of predilection is over the dorsolateral aspect of the frontal lobe. Perhaps because the patient lies recumbent there is a tendency for the pus to pass backward, and a secondary site of predilection is over the parietal lobe. Pus situated here may be connected to the frontal site via a narrow isthmus. It may extend down into the sylvian fissure but usually spares the temporal and occipital regions. Pus may also collect between the hemispheres, by extending directly from the frontal sinus or secondarily by extension of pus from a dorsolateral empyema. Such mesial or parafalcine empyemas may be unilateral or bilateral. Empyemas involving the base of the cranial cavity are uncommon, though a thin film of pus may collect over the orbital plates, and Hitchcock and Andreadis[2] (1964) reported a prepontine subdural empyema secondary to osteomyelitis. When the primary infection is in the ear, subdural pus tends to collect above the tentorium cerebelli rather than in the posterior fossa.[2]

Communicating veins pass from the cerebral veins to the venous sinuses, crossing the subdural space as they do so. Connections exist between the intra- and extracranial veins, passing either through foramina in the calvarium, or making connection with the veins of paranasal sinus mucosa.[3]

Etiology and Pathogenesis

Frontal extradural empyemas are mentioned less frequently than subdural ones, though it appears that small ones are fairly common.[4, 5]

The presence of pus in the subdural space has been termed purulent pachymeningitis, pachymeningitis interna, meningitis circumscripta, phlegmonous meningitis, intradural abscess, subdural abscess, and subdural empyema. The term subdural empyema is favored, as it denotes the presence of pus in a preformed space or potential space.[2, 6]

The majority of cases of extra- or subdural empyema follow purulent infections of the paranasal sinuses or middle-ear and mastoid air cells. Frontal sinusitis is now the commoner of these,[2, 7, 8] occurring either alone or as part of a pansinusitis. It is unilateral in more than half the cases.[9] Intracranial infection follows acute or acute exacerbation of chronic sinusitis more often than it does chronic sinusitis. Twelve of 14 cases reported by Kubic and Adams[6] followed paranasal sinus infection, 1 followed otitic infection, and 1 was metastatic from the lungs. Of the 33 cases reported by Schiller, Cairns and Russell[10] 23 followed paranasal sinus infection, 6 were due to primary otitic disease, and 4 were metastatic. Twenty-six of the 29 cases reported by Hitchcock and Andreadis[2] followed paranasal sinus infection, 1 followed otitic infection, and 1 was metastatic. Uncommon causes of subdural empyema are penetrating and nonpentrating wounds,[11] surgical craniotomy, skull fractures, infection of a closed preëxisting subdural hematoma,[12, 13] metastatic infection, usually from a primary pulmonary focus, rupture of an intracerebral abscess, or by extension of leptomeningitis. It has been suggested that the ratio of subdural empyemas to brain abscesses is increasing.[2, 14]

Though infection from the frontal sinus may sometimes extend inward by direct extension from a patch of ostcomyelitis,[10, 15] an alternative explanation was propounded as long ago as 1893 by MacEwen.[16] While accepting that such extension of infection from the sinus, via bone, to the intracranial structures may sometimes occur, this author also wrote that:

> "Leptomeningitis and cerebral abscess may form independently of a visible tract of inflammation spreading inward from the initial focus of irritation outside the cranial cavity. In such cases the pathogenic cause has been conveyed through the vascular system by direct extension from some source of infection to the meninges or brain. This may be due to thrombosis extending through the veins into the pia or the brain; or the veins may become blocked by a localized disintegrating thrombus, portions of which, containing pathogenic organisms, may be carried inward by the reversed blood stream. The sinus and intracranial veins lack valves, and the current of blood in them can be reversed on occlusion occurring at one point."

This mechanism gained strong support from the studies of Courville,[9] who pointed out that it is uncommon for a fistula to be demonstrable between the frontal sinus and an underlying empyema. Thrombophlebitis of the communicating veins has been shown postmortem, and this provides an explanation for the occurrence of extradural empyema, subdural empyema, superior sagittal sinusitis, cortical thrombophlebitis, or intracerebral abscess, either in isolation or in various combinations. An infected leptomeningitis occurs less commonly than subdural empyema from this cause, though a focal noninfected patch of arachnoiditis under a subdural empyema is common[9]; this accounts for the usual finding of pleocytosis in the spinal fluid. The occasional occurrence of spreading infection of the diploë or of an orbital abscess can also be explained by such extension along veins, and retinal thrombophlebitis has been reported.[17] In longstanding cases an encapsulating membrane forms which, like the membrane of a subdural hematoma, is thick under the dura and thin over the arachnoid. In places the dura may be bound to the arachnoid by an opaque yellow lamina containing fat granule cells.[10] Epicranial abscesses occur rarely, though epicranial edema may overlie an extradural empyema, that is, the so-called Pott's puffy tumor.

It would be difficult to invoke osteomyelitis of the skull as an etiologic factor in the development of an extradural empyema in our patient, but assuming his initial infection was in the frontal sinus, spread via the communicating veins could account satisfactorily for infection of both his extradural and subdural spaces. The prolonged prodromal illness, lack of roentgenologic evidence of sinusitis, and sterility of the subdural pus are consistent with the dampening effect of tetracycline and subsequent antibiotics. It would appear from a comparison of the second and first arteriograms that the subdural empyema developed acutely following drainage of the extradural pus. However, the presence of a significant membrane proves that it was relatively old. The marked shift of the anterior cerebral artery (figure 5) may be explained by increase in the quantity of subdural pus, change in position of the pus, or cerebral edema.

Organisms

Streptococci are the commonest organisms in these cases. Schiller, Cairns and Russell[10] grew the streptococcus in 24 out of 27 positive cultures; they accounted for 10 out of 15 positive cultures reported by Stern and Boldrey,[14] though sinusitis was the primary infection in only 5 of their cases; Hitchcock and Andreadis[2] found that the streptococcus was the commonest organism isolated among their 29 cases and that it was anaerobic in half of them. A mixed flora of 2 or more organisms was found in 5 of their cases. Other organisms are staphylococcus, pneumococcus, *E. coli*, clostridia, Proteus, and fusospirochetes. A case of actinomyocosis has been reported.[18]

Clinical Features

The typical patient is an adolescent or young adult male who suffers from an acute, or acute exacerbation of chronic, frontal sinusitis. Though the frontal sinus is rudimentary, cases of extra- or subdural empyema may originate from this site in children.[2, 3] Local signs of extension of the infection may appear, such as edema of the skin overlying the sinus or in the periorbital regions.[6, 9, 19, 20] The formation of an extradural empyema may not be obvious, since the malaise, fever, headache, leukocytosis, and other clinical features may be ascribed to the primary disease. Failure of the patient to respond to appropriate treatment or the development of further complications points to the correct diagnosis.

Subdural empyemas can occur alone or in association with extradural pus. They are more dramatic in their evolution and more serious in their import. The patient almost invariably develops a unilateral or generalized headache, and his condition deteriorates abruptly, with clouding of the sensorium and confusion.[2, 9, 15] Neck stiffness and other signs of meningeal irritation are common, and jacksonian convulsions frequently follow.

The clinical picture which develops depends upon the anatomic site of the empyema. When it is dorsolateral, seizures usually begin in the hand and spread to involve the face; the eyes and head may deviate to the contralateral side due to cortical irritation, and spread to the lower limb occurs later. Hemiparesis tends at first to be brachiofacial in distribution; an almost complete, flaccid hemiplegia may develop within 24 hours,[2] and the eyes then either deviate away from the paralyzed side, or the patient is unable to look toward the paralyzed side.[15] The hemiplegia may show a remarkable completeness in the presence of only a thin film of pus, and this contrasts with the rarity of hemiplegia occurring as a complication of purulent leptomeningitis.[9, 10] Dysphasia is often a striking feature.[2, 15, 21] It is predominantly more expressive than receptive in type, at least in the early stages, and its occurrence in left-sided empyemas has been emphasized by many authors. An exception was Courville,[9] who stated that it was relatively infrequent and reported its presence in only 6 out of 27 cases suffering from a left-sided lesion. His study was retrospective, and the true incidence may have been higher than this. Hemihypesthesia may be demonstrable, but the patient is usually too sick to coöperate with more extensive testing. Hemianopsia is not a feature of dorsolateral empyemas. Bradycardia may be commoner with subdural empyemas than intracranial abscesses,[9] and this may serve as a differentiating point between the 2 conditions.

In patients with parafalcine empyemas the signs follow a different pattern of evolution. The general symptoms at the time of onset are similar to those occurring with dorsolateral collections of pus, but jacksonian seizures begin in the foot, whence the march proceeds centripetally. Weakness involves the lower limb, often with sparing of the face; dysphasia may occur from involvement of the secondary speech area anterior to the paracentral lobule, as well as hemianopsia from involvement of the calcarine cortex.

When the primary source of infection is in the ear, extradural collections of pus are not uncommon, and brain abscesses may occur in the temporal lobe or cerebellum without more than a purely local meningitis. With the supervention of a subdural empyema the patient may show excessive somnolence, and homonymous hemianopsia may be present.[10]

Edema of the underlying brain is common in these patients, and uncal or tonsillar herniation may occur.[10, 22, 23] Unequal pupils may appear, but they are not a reliable indicator of the side of the lesion.[15] A dilated pupil appeared on the contralateral side in 2 of Courville's patients.[9] Changes commonly occur in the optic disks; they take the form of blurring of the margins with filling of the cups and venous engorgement, rather than marked papilledema. Leukocytosis is almost invariably present in the peripheral blood; this, also, may be a point of differentiation from brain abscesses, in which leukocytosis is absent in a large proportion of cases.[4] Levels average 20,000 cells and may be in excess of 30,000.[9, 19, 20]

Diagnosis, Treatment and Prognosis

Spinal fluid. The spinal fluid findings vary with the stage of the disease. The pressure is elevated, and there is usually a mild to moderate pleocytosis, consisting predominantly of polymorphonuclear cells. The protein content is elevated, but sugar, smear, and culture are normal. Cell counts varied between 15 and 840, and polymorphonuclears between 0 and 90 per cent in one series,[6] and cell counts as high as 4,880 have been reported.[2] However, in 8 (35 per cent) there was almost normal spinal fluid in this latter series.

In the absence of lateralizing signs valuable information may be obtained from the discovery of a displaced calcified pineal body, an asymmetric echoencephalogram or electroencephalogram, or an abnormal brain scan. However, angiography provides the greatest assistance to the surgeon in locating areas of suppuration. As in the case cited above, this may need to be done on more than one occasion.

Radiographic features. The angiographic pattern of epidural and subdural empyema is, at least in early lesions, identical with the more common hematomas. The diagnosis depends upon the demonstration of displacement of arteries and veins away from the inner table of the skull, the avascular space of Löhr. Certain findings make possible the angiographic distinction of extradural from subdural collections: (1) inward displacement of the meningeal artery, (2) downward displacement of the superior sagittal sinus, and (3) rupture of a meningeal vessel with extravasation of contrast material. It is said that extradural hematomas are less apt to displace the anterior cerebral artery; that they do not extend over a large area; that they are more likely situated in the temporal fossa; and that the border of the hematoma against the brain is not smooth.[24-28]

If extra- or subdural empyemas persist a capsule forms, and a richly vascularized zone develops in the bordering brain tissue.[29] This explains specific findings in extracerebral empyemas described by several authors.[23, 29, 30] Ferris and Ciembroniewicz[23] list 3 angiographic features which are thought to be diagnostic of subdural empyema: (1) "vascular pooling" in the dural wall in AP and/or lateral views, (2) an avascular space, semilunar in shape, in the lateral view, and (3) irregular detachment of vessels from the inner table in the AP view, giving a scalloped appearance to the inner margin of the empyema. None of these findings was present in our case.

There is no controversy regarding the mode of treatment for extradural and subdural empyema. Immediate evacuation of the pus collections and the administration of antibiotics are essential. Though more extensive exposure has been advocated,[5, 31, 32] the aim can usually be accomplished by strategically placed burr-holes.

If the patient's clinical course is unsatisfactory, a persistent search for undisclosed collections of pus is of paramount importance. In the case described it seems likely that the extradural and subdural collections coëxisted, though the former must have been more advanced initially in view of the angiographic findings. In retrospect, it would have been better to have opened the dura at the time of the second operation when nothing was disclosed extradurally. Schiller, Cairns and Russell[10] thought that the hazard of spreading infection by opening the dura in the presence of extradural pus was minimized by penicillin. Topical use of this antibiotic is now avoided because of its possible epileptogenic effect.[33] It is of interest that smears and cultures of the subdural pus were negative for bacteria, attesting to the effectiveness of appropriate antibiotics in limiting the infection.

The outlook for these patients has improved since the use of brain scans and angiography to locate the pus has become routine. Nevertheless, as recently as 1964 the operative mortality for subdural empyema was 27 per cent.[2] The main cause of failure was undetected or inadequately drained loculations. It is likely that surgical intervention promotes the formation of such loculations.[34] They may extend along the falx between the hemispheres or on the tentorium. Failure to treat unsuspected coëxisting brain abscess or abscesses is another cause of ultimate demise.

Adequately treated patients with interhemispheric subdural collections do better than those whose disease involves the convexity of the brain.[20] In the latter instance, permanent residual aphasia, paresis, or seizures may occur in survivors.[35]

Summary

The etiology, clinical and diagnostic features, and treatment of extra- and subdural empyemas are discussed in the light of an illustrative case, the course of which appears to have been modified by previous antibiotic therapy.

The outlook for these patients has improved since the use of brain scans and arteriography has become routine. The radiologic features of extra- and subdural empyemas are discussed. If the patient fails to respond to the accepted treatment of surgical drainage plus antibiotics, repeated arteriography should be undertaken to seek further collections of pus.

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(*Continued on page 516*)

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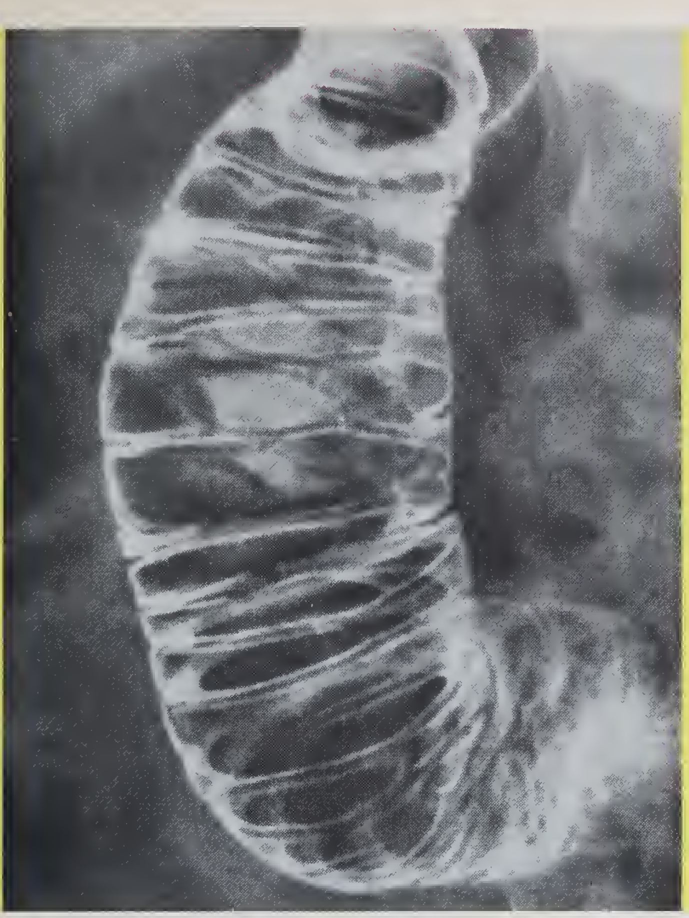

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President's Page

STUDENTS, ONE BY ONE

No dedicated teacher of medicine, or of the arts and sciences, can fail to be disturbed by the waves of student unrest which have plagued our colleges and universities. That these demonstrations have not been the expressions of a few outsiders and beatniks was indicated in a *Life* magazine editorial which revealed that 80 per cent of the persons participating in the massive sit-in demonstration at the University of California on December 3, 1964 were properly registered students. Approximately half of the undergraduates arrested had better than 'B' average records at the school. No, the revolts were symptomatic of a seething unhappiness in the student bodies. Was this due to the failure of institutions to recognize the identity of each of their students as individuals?

Unfortunately, teaching for its own sake has become eclipsed by increasing emphasis upon research. Dr. John Weaver, Dean of Faculties at Ohio State University, quoted a memorandum received by a university vice president for medical sciences which read: "There is no merit to be gained in teaching; you are expected to bring in 20 to 50 per cent of your salary through grants; if you are unwilling to do your share to make this institution internationally famous through research you are invited to leave." Noting that Federal expenditures for research and development had increased from 74 million dollars in 1940 to 15 billion dollars in 1964, the Carnegie Foundation for the Advancement of Teaching indicated that the Federal Government has had a tremendous influence on the deterioration of teaching and, in fact, upon the teacher shortage; to quote: "The able researcher, through publication, gains a national reputation. But the able teacher is rarely known, as a teacher, beyond his own college or university. Good teaching is not only a relatively private performance, but it resists measurement." A man distinguished for his electrifying discoveries may be a poor instructor. His time is so occupied by conferences and lecture tours that he passes the student classes down to a subordinate who, in turn, relegates this responsibility to men in the still lower echelons. That good students will no longer tolerate such neglect was emphasized by a recent student revolt in one of our most distinguished medical schools.

No one can doubt the importance of meaningful scientific research. Laboratory investigation and student instruction, however, are often poles apart. Dr. Weaver pointed out that the Latin word from which we derive our verb 'to educate' has the meaning 'to lead forth.' When that leadership in the form of truly effective pedagogy is lacking, perhaps it is our attitude toward the students rather than the attitude of the students toward the universities which needs reappraisal.

William S McCune

Editorials

Transplantation of the Heart—Dissenting Notes of a Cardiologist*

"The *heart* has its reasons which the reason does not know."—*Pascal*

"My *heart* leaps up when I behold
A rainbow in the sky."—*Wordsworth*

Even in 1968 it is difficult to talk objectively of the heart, that most ubiquitous of poetic images. The heart has long been regarded as the essence of man, the seat of love and affection. Words such as sweetheart, heartbroken, heartless, heartfelt, soft-hearted, hard-hearted, etc. are commonplace in our vocabulary. Some "eat their hearts out," others "wear their hearts on their sleeves," still others are asked to "have a heart," while a few have "hearts of gold."

With all these preconceptions to color our reactions, we learn that a 41-year-old Dallas housewife and mother of 5 children has received the world's twenty-first heart transplant. How thrilling, we cry! It seems that a therapeutic panacea is just around the corner. We breathe a sigh of relief and the thought crosses our minds that some day we ourselves might be lost but for the grace of cardiac transplants. Would that this were so! In the meantime, both the medical profession and the communications media have a heavy responsibility to counsel the public rather than to excite or mislead. Advances in biology and medicine tend to have wide social implications. Public interest is both understandable and desirable. However, each technological step exacts a price, and we should be prepared to ask how much is likely to be gained from the effort expended. Where does each advance fit into the general economy of our medical effort? Our resources are not unlimited, and we must accordingly establish a system of priorities governing the direction of our efforts. There is concern that the dramatic impact of cardiac transplantation may divert time, personnel, and funds from areas of clinical investigation that are potentially more important to human welfare.

The number of people who need new hearts is much greater than the current or projected supply. This number appears to be well beyond the ultimate potential of cardiac transplants. When the demand far exceeds the supply of organs, the haunting question of distribution arises and must be faced before it becomes a reality. Cardiac transplantation may ultimately prove to be biologically sound but therapeutically less important. Professional time, personnel, and money are basic elements in medical care. Use of these elements should be questioned when large numbers of skilled professionals become absorbed in the care of small numbers of sick people. Such an application of resources is undesirable as long as the role of medicine is the treatment and prevention of disease in the entire population. Dr. Lewis E. January, President of the American Heart Association in 1967, said, "I think whatever happens to heart transplants over the years, it's always going to be peripheral to the main problem and that is sufficient investigation and basic research, so that we can prevent the type of heart disease that we are talking about."

We should unhesitatingly criticize any group that performs a cardiac transplant without committing itself to the advancement of research in this field. Transplantation of the heart can easily degenerate into a status symbol designed to improve the image of an institution or attest to the expertise of a surgeon. The temptation is great, but it must be resisted at all costs if professional integrity is to be preserved. The ethics involved becomes a part of the complex question of the ethics of human experimentation. A number of well-qualified groups both here and abroad were already addressing themselves to this sensi-

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* Summary of remarks made in a Panel Discussion on "The Ethics and Morality of Heart Transplants" during the Tenth Biennial Cardiovascular Seminar held in Miami, Florida, June 6–8, 1968.

Opinions expressed in contributions to the Editorial Section are those of the writers and do not necessarily reflect the views of the Medical Society of the District of Columbia

tive problem before transplantation of the human heart became a reality. The efforts of these groups continue, but until they can provide us with acceptable guidelines we must make every effort to impose upon ourselves certain elementary disciplines. Accordingly, the Board of Medicine of the National Academy of Sciences has proposed that cardiac transplantation should be carried out only in institutions in which the following criteria can be met:

"1. The surgical team should have extensive laboratory experience in cardiac transplantation and should have demonstrated not only technical competence but a thorough understanding of the biological processes that threaten functional survival of the transplant, i.e., rejection and its control. Investigators skilled in immunology . . . should be readily available as collaborators in the transplantation effort.

"2. As in any other scientific investigation, the overall plan of study should be carefully recorded in advance and arrangements made to continue the systematic observations throughout the whole lifetime of the recipient. The conduct of such studies should be within an organized framework of information exchange and analyses.

"3. Rigid safeguards should be developed with respect to the selection of prospective donors and the selection of prospective recipients. An independent group of expert, mature physicians, none of whom is directly engaged in the transplantation effort, should examine the prospective donor. They should agree and record their unanimous opinion as to the donor's acceptability on the basis of the evidence of crucial and irreversible bodily damage and imminent death. Similarly, the prospective recipient should be examined by an independent group of competent physicians and clinical scientists including a cardiologist and an expert in immunology. The consultant group should also record their opinion as to the acceptability of the recipient for transplantation on the basis of all the evidence including the presence of far-advanced, irreversible cardiac damage and the likelihood of benefit from the procedure."

Certain impressions emerge when cardiac transplants are considered in the light of the foregoing comments. Research granting-agencies should confine their support for transplantation of the heart to the limited number of institutions that qualify under criteria such as these recommended by the National Academy of Sciences. The Committee on Ethics of the American Heart Association has endorsed these criteria and expressed the view that "only a small number of studies, carefully performed, are needed to provide the scientific information that will determine the future role of cardiac transplantation in man. . . ." The public should be aware that at the present time cardiac transplantation is not justified as a form of treatment but only as an area of clinical investigation. The National Academy of Sciences stated that transplantation of the heart "must be clearly viewed for what it is—a scientific exploration into the unknown, only the very first step of which is the actual surgical feat of transplantation of the organ."

Although the ethical issues of cardiac transplantation relate chiefly to the general subject of human experimentation, certain problems arise specifically from the use of the heart itself. First, the donor must die when a heart is transplanted. This is in contrast to removal of a paired organ such as the kidney in which the donor's life continues. A recent article in the *American Journal of Medicine* pointed out that the continuing risk to a young adult following removal of a kidney was about the same as commuting 7 miles into an urban area in the United States by car. Second, if a transplanted heart fails to function, the life of the recipient cannot be salvaged. If a kidney transplant fails, the recipient's life can sometimes be maintained pending a second attempt. Third, the issue of cardiac transplantation is colored by the symbolic connotations alluded to in the opening paragraph. It is little wonder that cardiac transplantation has emerged as a spectacular surgical procedure that captures the imagination far more, for example, than liver transplantation which is technically as difficult and equally complex. But alas, the liver is not endowed with poetic imagery.

The legal problems are many, but among the most important are the issues of voluntary and informed consent and the definition of death. Doubt has been expressed as to whether the circumstances surrounding impending or actual death preclude an exercise of judgment necessary to give the type of informed consent required by law. Although this is a relevant consideration, I do not think the subject of consent is insurmountable. Consent for removal of organs for autopsy is commonplace and poses no special problem. Removal of the heart for transplantation is in many ways legally analogous, provided the donor is dead. The definition of death must therefore be examined closely. Definitions change, and there is little doubt that cessation of the heart beat as a criterion of death is an anachronism.

This problem was dramatically posed in 1929 by Luigi Pirandello in his play "Lazzaro." The action of the play took place in a small village in Italy where the people temporarily lost their simple faith because a man revived after his heart had stopped and had no recollection of having been in paradise.

When someone is seen walking down the street engaged in lively conversation there is no dispute that he is alive. When a corpse has been lying in a morgue refrigerator for several days there is no dispute that it is dead. The extremes are easy to define, but delicate judgment is required to determine just when the transition from one state to the other has occurred. Such a judgment must be made if hearts are to be removed quickly enough to be of use as transplanted organs. Some physicians would not want to be called upon to make such a decision, while others would feel competent to do so. Both points of view are understandable, but—like the legal issue of consent—I do not believe that the medical definition of death will prove to be insurmountable. It must be firmly stated, however, that the decision of when to remove the heart of a donor should not rest with the physician who is in charge of his medical care. British hospitals already require that the physician responsible for the care of a prospective donor cannot at the same time be a member of the transplant team. The areas of responsibility defined by the National Academy of Sciences support this view.

The dramatic impact of cardiac transplantation is undeniable. However, at the present time the public must realize that the procedure cannot be regarded as an established form of treatment; that it is justified only as a scientific investigation; and that, although complex legal and ethical issues arise, the essence of the problem is the determination of just where in the general economy of our medical effort such a procedure legitimately fits. We are obliged to consider the wisdom of allocating disproportionate amounts of personnel, time, and money to the care of relatively few people at the potential risk of neglecting the welfare of the many.

Joseph K. Perloff, M.D.
Associate Professor of Medicine,
Georgetown University School of Medicine

Planned Blood Therapy

Today the various components of blood can now be specifically separated and used in planned blood therapy by the physician. This has been made possible through refined techniques which permit the separation of the various components of whole blood. The result is a more efficient and precise means of meeting the patient's need.

It has been estimated that whole blood is necessary in only 10 to 50 per cent of the transfusions administered in medical practice. However, a much larger percentage of whole blood is used in the average hospital. After severe hemorrhage the use of whole blood is necessary. In anemic states which do not require volume expansion packed red cells are more desirable, because the use of whole blood in these cases could lead to circulatory overload. The use of packed red cells in such instances will then make plasma available for other uses. The use of packed red cells also decreases the number of transfusion reactions due to a plasma factor. For instance, some febrile reactions in patients who have had repeated transfusions are due to the reaction of the leukocyte antibodies of the recipient with the leukocytes of the donor. The use of packed cells free of white cells will prevent these reactions.

The Committee on Blood of the Medical Society of the District of Columbia, therefore, makes a plea to all physicians to review their transfusion practices and urges them to use specific components when possible and, in particular, packed red blood cells when applicable.

Vernon E. Martens, M.D.
Chairman, Committee on Blood Banks

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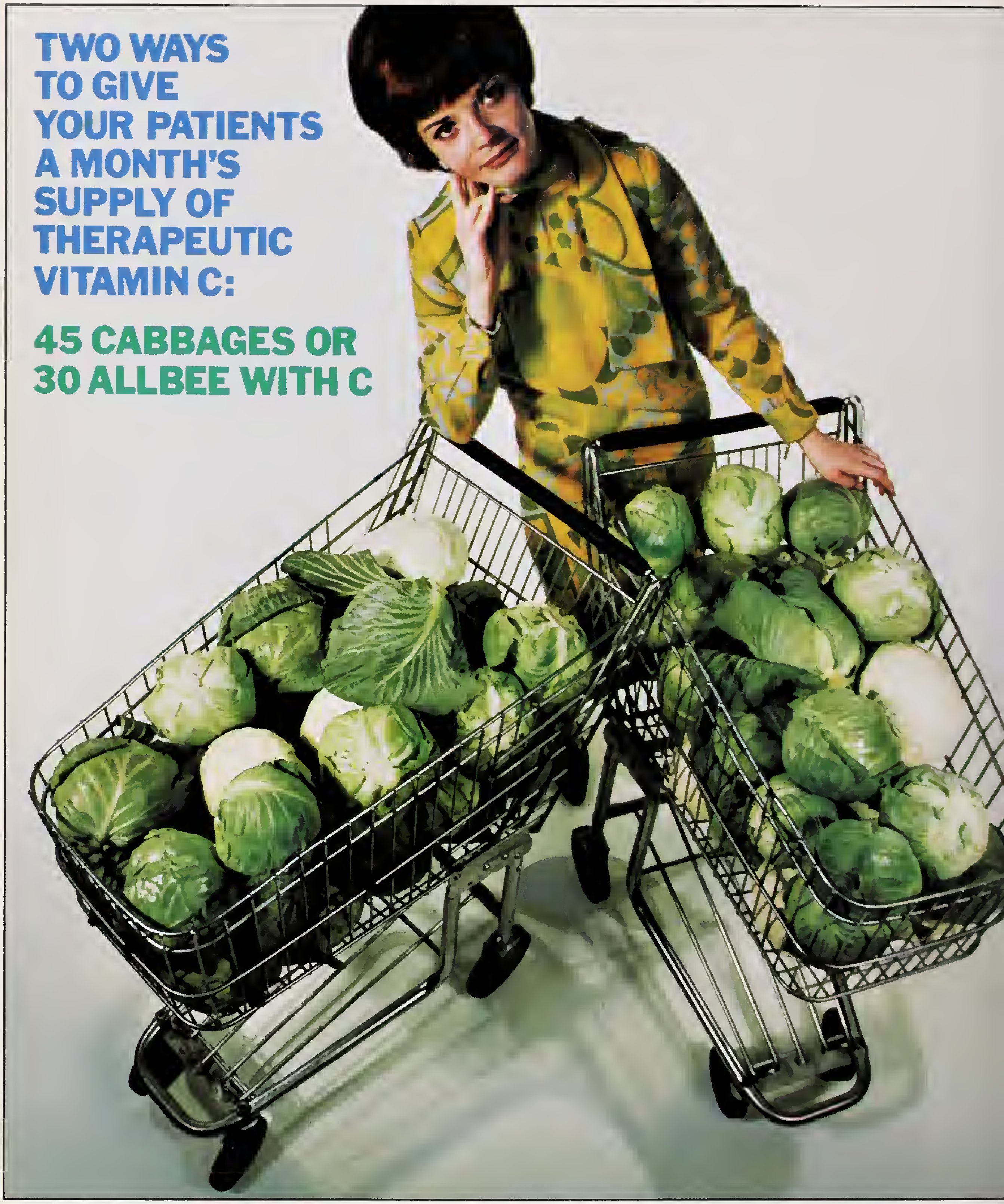

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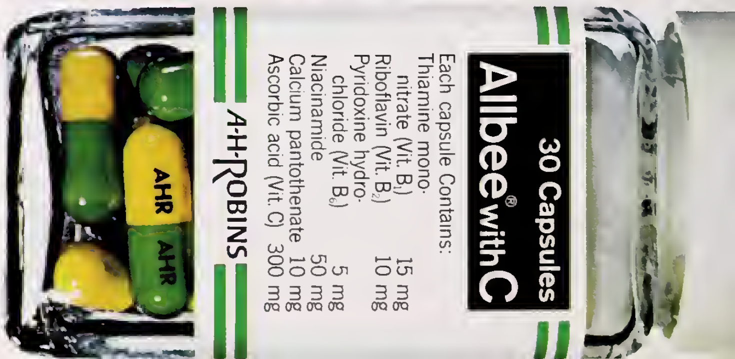
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Report of the Delegates to the AMA House of Delegates

San Francisco, Calif., June 16–20, 1968

THE OPENING SESSION of the meeting of the House of Delegates on Sunday, June 16, 1968 was interrupted by 2 men who slipped past the guards at the entrance and seized the microphone. One was a medical student from Stanford University, representing the Medical Committee on Human Rights. The other was a welfare worker and official of the Bay Area Poor People's Campaign. Rather than have them ejected, Dr. Walter C. Bornemeier, Speaker of the House of Delegates, allowed them to speak, which they did in turn and were then led out. Though there were rumors of further interruptions, none occurred and the incident which did occur received much more attention and publicity than it warranted. A written statement outlining the objectives of the Medical Committee on Human Rights was read to the House later as a matter of information, but it contained nothing which was not already quite well known and appreciated by the delegates present.

Once it got to work the House of Delegates considered a total of 143 items of business, including 84 resolutions and 59 reports from Councils and Committees.

In a history-making decision the House reaffirmed the previous position of the Association in strong opposition to discrimination against applicants for membership and approved a resolution that the By-laws be amended to provide for punitive action against component or constituent associations in which discrimination is proven to exist.

Relative to health care costs and financing, the House approved a report of the Committee on Health Care Financing which stated that recent advances in health care have served to vastly increase the demand for and the cost of health care services. Adequate health care should be available to all who need it, and methods of financing must aid all individuals to achieve it. Voluntary programs of financing must accommodate to continuing changes and growth.

Proposals under study include a community fiscal agency for better services, income tax credits for premium paid for adequate health insurance, and methods of promoting maximal participation in voluntary comprehensive health programs.

A resolution that "the principles of graduated income tax credits for premiums paid for adequate health insurance be adopted as approved policy of the AMA" was adopted.

Another resolution adopted urged all state and local societies to act swiftly and firmly in all instances of known exploitation and excessive charges for health care in their jurisdictions.

A report of the Board of Trustees, which was adopted, will cause a technical evaluation of the potentialities of the AMA in the field of economics of health care to be carried out promptly by an advisory group of competent, nationally recognized economists.

Resolutions calling for voluntary insurance coverage for outpatient X-ray and laboratory services acceptable to the hospital and its medical staff wherever performed prior to a scheduled hospital admission and for the adoption and authorization of the use by insurance carriers of standard insurance forms in conjunction with a single hospital admission were adopted.

Two resolutions concerning comprehensive planning were combined to state the House's will that "medical societies encourage the organization of local health planning councils without the assistance of HEW grants."

The House also expressed its will that "the society should use some mechanism such as a nonprofit corporation or other entity, separate from the society itself and served by an independent administrative staff, where possible, to avoid direct operational involvement of the medical society as a body."

A Council on Medical Service report adopted by the House pointed out:

> "There are no easy, black-or-white solutions to this dilemma. With government increasingly involved in research and experiment in the delivery of health services and payment for care, we cannot ignore government programs. On the other hand, the multiplicity of programs and approaches at the national, state and local level precludes blanket endorsement of any given level of involvement. The Council therefore recommends that this House make it clear that medical societies may, after mature consideration, legitimately take part in such programs ... and that it should trust to the good sense of the individual state and county societies to recognize the serious import and the need for careful study before deciding on such a step."

To strengthen the AMA's continuing liaison with the Federal Government on matters of health legislation, the House adopted a statement that because "the private practicing physicians are the best qualified group in matters pertaining to health services," the AMA should "request the Secretary of HEW to continue to solicit the advice

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Side effects include drowsiness; dizziness; nausea; excitement; hypotension; fine tremor; jitteriness; weakness; headache; heartburn; anorexia; increased perspiration; incoordination; allergic-type reactions manifested by skin rash, swelling of face and tongue, itching; numbness and tingling of limbs, including peripheral neuropathy; activation of schizophrenia which may require phenothiazine tranquilizer therapy; epileptiform seizures in chronic schizophrenics; temporary confusion, disturbed concentration or, rarely, transient visual hallucinations on high doses; evidence of anticholinergic activity, such as tachycardia, dryness of the mouth, blurring of vision, urinary retention, constipation; paralytic ileus; jaundice; agranulocytosis.

Careful observation of all patients is recommended. The antidepressant activity may be evident within 3 or 4 days or may take as long as 30 days to develop adequately, and lack of response sometimes occurs. Response to medication will vary according to severity as well as type of depression present. Elderly patients and adolescents can often be managed on lower dosage levels.

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and counsel of those practicing physicians designated by their medical associations at the local, state and national level in formulating future plans and programs and in modifying existing programs, in order to provide the best quality health care to the people of the United States."

Three resolutions were introduced objecting to the government's support of group practice, particularly the prepayment type. They were combined into one substitute: "Resolved, that the AMA continue to espouse the private, fee-for-service practice of medicine; and be it further resolved that the AMA strongly disapproves of the provision of funds by the Federal Government for subsidizing any one form of organization of medical practice."

Adopted was a resolution that an attempt be made to change the terminology in the Medicare "Exploration of Benefits" forms to eliminate existing confusion and criticism. Reaffirmed were the previous policies requesting that all efforts be made to eliminate recertification requirements under Medicare and that the laws and regulations relative to PL 89–99 be amended to permit direct billing to all Title 19 recipients.

The Board of Trustees was directed to take appropriate action seeking the establishment of a separate Department of Health headed by a doctor of medicine.

In response to a report from the Board, a Council on Health Manpower was created by the House to assume the functions and responsibilities of the former Committee on Health Manpower and the former Commission on Relationships between Medicine and Allied Health Professions and Services.

The House approved a Board report reviewing studies of the status of foreign medical graduates in the United States and directing the new Council on Health Manpower to take the steps necessary to complete plans for the organization and financial support of a commission, to be established outside of government, to coördinate fully all of the issues and activities involving foreign medical graduates.

A Board of Trustees' report was approved which urged that "the constituent and component societies of the Association make every effort to insure that additional financial support for medical schools be obtained from all available sources. The Board and the Council (on Medical Education) further urge that all physicians individually recognize their debt as graduates of medical schools . . . by contributing regularly and generously to the financial support of medical schools through the 'Fund for Medical Schools' project" of the AMA-ERF and other appropriate channels.

Another resolution which was referred to the Board related to the ruling of the Joint Commission on the Accreditation of Hospitals (JCAH) with respect to the use of externs, under proper supervision, for recording history and physical examinations on patients' charts in non-university affiliated hospitals.

The House approved a statement on infant mortality prepared by the Committee on Maternal and Child Welfare of the Council on Medical Service and submitted by the Board. The report included a number of reccommendations, among which were these:

> "Recognizing the fact that unfavorable environmental and socio-economic factors as well as medical factors are involved in infant mortality, the medical profession should support all constructive community efforts for the improvement of living conditions among the needy.
>
> "The AMA should inaugurate and support programs of health education, including good maternal and child health practices, family life and sex education, and the appropriate use of health care resources.
>
> "The AMA should give impetus to broad and inclusive programs for the care of the unwed mothers.
>
> "The AMA should provide leadership in a program to obtain national uniform registration statutes for births and deaths . . . promote efforts through the World Health Organization and other authoritative bodies to bring about uniform registration requirements and . truly comparable statistics . . . and support educational programs directed toward physicians concerning the value of accurate uniform reporting.
>
> "State and component societies should evaluate existing maternal and child care resources and take leadership in the expansion and development of programs for delivering services in locations found to be deficient."

The report urged that, where feasible, "perinatal death studies be conducted on a community-wide basis or by a hospital staff or medical group for the purpose of reducing infant mortality and providing educational programs"; it also recommended that the AMA "stimulate research into the cause and possible prevention and control of 'crib deaths' of infants."

A Judicial Council report was adopted on "Ethical Guidelines for Organ Transplantation." One of several important guidelines was: "When a vital, single organ is to be transplanted, the death of the donor shall have been determined by at least one physician other than the recipient's physician. Death shall be determined by the clinical judgment of the physician. In making this determination, the ethical physician will use all available, currently accepted scientific tests."

A Council on Medical Service report on automated multiphasic screening, approved by the House, pointed out that tests used in "a multiphasic screening program should be relatively simple to administer, easy to interpret, relatively inexpensive and require little time to perform." All such screening programs should be subjected to the 5 criteria suggested by the former National Commission on Chronic Illness: reliability, validity, yield of information, cost, and acceptance by physicians, individual laymen and the community.

The House resolved, in connection with proposals for a national compendium of approved drugs, that "present quality standards imposed voluntarily by the American pharmaceutical industry should not be abridged or usurped by any Federal agency, and . . . any drug compendium devised should be made available solely as a reference source of information."

The Board was directed by the House "to evaluate the practicality and feasibility of establishing a national health data banking system and to evaluate other aspects of data processing related to the practice of medicine."

The House resolved to continue AMA "efforts to safeguard and inform the medical profession and the public with respect to the treatment of obesity."

The AMA's policy on chiropractic was reconfirmed, and state and local medical societies are urged "to formally adopt the AMA Policy Statement on Chiropractic, or a somewhat similar expression" and "to alert the general public to the health hazard posed by the cult of chiropractic."

By adopting a Board report the House voted to "participate in a program of planning and action with other organizations concerned with architectural barriers to the handicapped" and requested state and county medical societies to encourage their members to become active in helping to alleviate such barriers.

Noting that new Standards for Hospital Accreditation are under way by the JCAH, the House resolved that the Board "direct the AMA Commissioners of the JCAH to arrange for the tentative draft of the new standards to be submitted to individual state associations for comment before final adoption."

The House again urged "that adequate representation of the medical staff on the voting membership of the hospital's governing body is the most effective mechanism for assuring a working communication, provided that the physicians serving on the governing board be nominated by the medical staff of the hospital."

Regarding use of paramedical personnel, the House recommended to the JCAH, American Hospital Association and its state and local chapters, and to hospitals and other related facilities that the practice of medicine "be preserved as the responsibility of the physician and that paramedical personnel not be placed in the position of practicing medicine whether by consent, design or contract," and it recommended to the state and county medical societies and hospital staffs that they "call attention to the dangers of contractural agreements which remove the services of paramedical personnel from the supervision of the physician."

A Board of Trustees' report informed the House about the Association's public relations counsel, Mr. Philip Lesly. In a brief statement he pointed out the many problems facing the profession and his approach to them. He urged that as a profession our public relations must be (1) practical, (2) positive, (3) anticipatory, (4) selective, and (5) augmented by the voices of respected laymen.

The financial status of the Association was discussed in a report from the Board. A substantial increase in the budget is anticipated. This is because of the greatly increased cost of providing normal services due to the pressure of inflation and to the sharp rise in the cost of major items such as paper and printing.

The effect of taxes due to IRS regulations on the approximate 13 million dollars gross annual income from advertising is not known as yet but is certain to be considerable. No increase in dues is recommednded at this time and the Board will gather all necessary information before reaching such a decision.

The House adopted a resolution urging that expenditures of the Association be reduced and that the Board continue to cut back, consolidate, and eliminate such activities as are not productive.

A number of amendments to the By-laws were adopted. These were mostly of a procedural nature.

A new "Standard Code of Parliamentary Procedure" by Sturgis is to be studied along with the newly revised "Robert's Rules of Order" to determine which will be best suited to govern the proceedings of the Association.

Dr. Gerald D. Dorman, of New York, was named President-elect by acclamation. Dr. Carl A. Lincke, of Ohio, was similarly elected Vice President. Dr. Bornemeier, of Illinois, and Dr. Russell B. Roth, of Pennsylvania, were reëlected

Speaker and Vice Speaker, respectively, of the House, also by acclamation.

Three Trustees were elected for their first term: Dr. Raymond T. Holden, District of Columbia; Dr. John M. Chenault, Alabama; and Dr. John R. Kernodle, North Carolina. Dr. L. Otis Simenstad, Wisconsin, was elected to succeed himself.

Your senior delegate, who was elected a Trustee through the whole-hearted support and help of our President, his fellow delegates, a number of fellow members, and our new Executive Director, is fully appreciative of the honor of the new position and wishes to take this opportunity to thank all of his fellow Society members for their confidence and support over the years and for the opportunity they have given him to act as their representative. He is deeply grateful.

Raymond T. Holden, M.D., *Delegate*
John J. Hughes, M.D., *Delegate*
Thomas Sadler, *Executive Director*

DOCPAC BRIEFS

or

A Congressman Endorses AMPAC/DOCPAC

At the Third Annual DOCPAC Banquet in the Medical Society's Building, May 15, 1968, Congressman Walter S. Baring, Democrat of Nevada, gave the following pertinent remarks:

"There is still another way you can play an important part in the shaping of our country and that is by participating in DOCPAC and AMPAC even more than you are now doing. I cannot stress participation in DOCPAC and AMPAC too strongly. Through these organizations you can help elect members to Congress who share your political thoughts and views . . . through your participation you can see to it that the members of Congress who think along your lines remain in office.

"I can personally vouch for the wonderful work AMPAC has done for me in my state . . . Nevada.

"I sincerely suggest that you become active in the party of your choice. . . . Get behind the candidate that you feel is best qualified and go all out for him.

"Become even more of an active participant in DOCPAC . . . AMPAC than you are . . . . Get your colleagues interested in becoming active participants. Heaven knows, we have enough apathy when it comes to the field of politics.

"Three pieces of medical legislation that have my complete support are the Regional Medical Programs, the Health Manpower Act, and the Hill-Burton Act.

"The extension for 5 years of grants initiated by the Heart Disease, Cancer, and Stroke Amendments to establish Regional Medical Programs will, I am sure, receive the wholehearted support of Congress. The tremendous strides made in these fields make it imperative that the work be continued.

Narcotic Don'ts for the Physician

Don't fall for "a good story" from a stranger claiming an ailment that usually requires morphine.
—The addict can produce bloody sputum and urine and simulate a bad cough or other symptoms. Make your own diagnosis and prescribe accordingly.

Don't give a narcotic prescription to another person without seeing the patient for whom the prescription is written.
—Diagnosis is difficult without examination. Look for addiction signs when suspicious. Diversion to addicts is a profitable business, as much as $3 for ¼ grain morphine sulphate tablet.

Don't prescribe narcotics on the advice that another physician has been doing so.
—Consult that physician or the hospital records whenever possible.

Don't leave signed prescriptions at the office for nurses to complete.
—Signed blanks are bad practice and many have been stolen by addicts.

"The Health Manpower Act, Title One, extends to June 30, 1973, the authorization of appropriations for grants to construct medical and dental teaching facilities. It provides for facilities to be used for teaching purposes when being used for research and related purposes in the sciences related to health or medical library purposes.

"Title Two of the act, Nurse Training, authorizes appropriations for construction grants of new facilities or rehabilitation of existing facilities for collegiate, associate degree, or diploma schools of nursing for the next 3 fiscal years.

"The Health Manpower Act deserves the full support of Congress, and I am sure it will receive same.

"The Hill-Burton Act calls for increased appropriations under Title Six and extends the period of these grants for 4 additional years through 1973. I have always been a supporter of this Act since its inception and will do all I can to assure its passage when it comes on the House floor."

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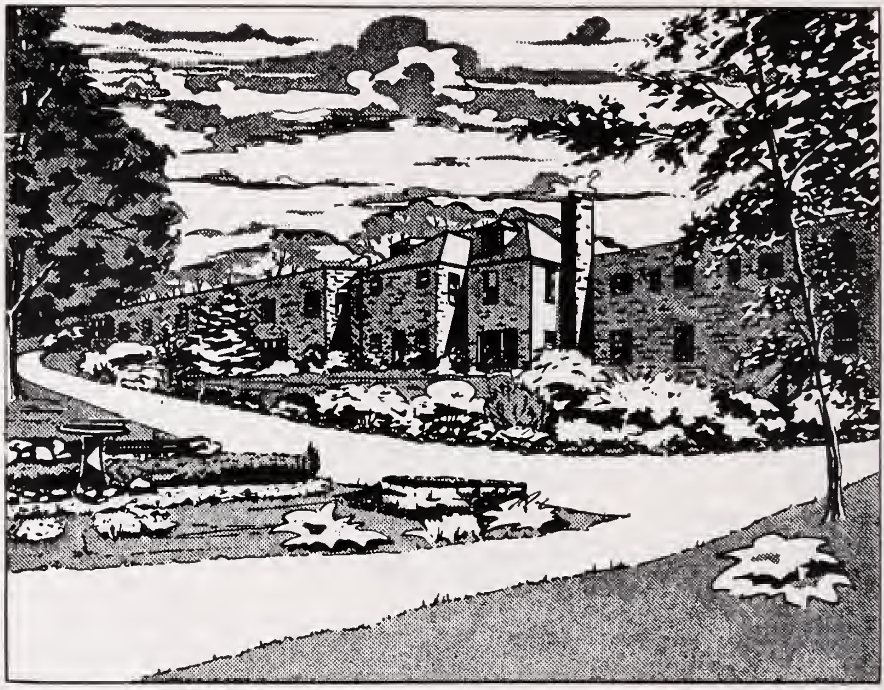
William H. Cooper, M.D.
Chairman, D. C. Political
Action Committee



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The Social Security Disability Program

How Recent Amendments Will Affect the Physician and His Patients

BERNARD POPICK*

PRIVATE PRACTITIONERS came under Social Security in 1965. In addition to earning retirement and survivors' protection, they may also have earned disability protection. Work credits could have been accumulated before 1965 through internships, residencies, military service, and other covered employment. This article discusses the disability program and how recent changes in the law affect the physician and his patients.

If you pay—or if you have ever paid—Social Security contributions on your earnings, you and your family may be covered by the Social Security disability program.

In the disability program, a person can receive monthly payments if he is disabled because of a severe physical or mental impairment which has lasted (or is expected to last) a year or longer.

The disability program was improved by legislation that became law early this year. As a result of an increase in benefits under the new Social Security amendments, monthly payments can amount to $189.90 for a disabled worker and $395.60 for a family. In forthcoming years maximum monthly benefits will be as much as $218 for an individual and $434.40 for a family.

To be insured for disability purposes, a person disabled when he is 31 or older needs to have worked under Social Security for at least 5 of the 10 years preceding the onset of disability.

For younger workers the amendments ease this requirement. A person who becomes disabled before his thirty-first birthday now needs Social Security work credits for only half the time between his twenty-first birthday and the onset of disability. Thus a person disabled at 26 needs only $2\frac{1}{2}$ years of credits—half of the 5 years between 21 and 26. A minimum of $1\frac{1}{2}$ years of credits is required.

Under the new amendments an additional group of people—disabled widows (including certain surviving divorced wives) and disabled dependent widowers of insured workers—can become eligible for disability benefits at 50 or older, even though they themselves never worked. (Widow's benefits are slightly reduced for each year she receives them before she is 62.) The disability must occur within 7 years of the spouse's death or, in the case of a widow caring for a child entitled to benefits, within 7 years after her mother's benefits end.

Social Security Disability Provisions at a Glance

- Benefits go to disabled workers under 65 and their dependents.
- A disabled worker needs a certain number of Social Security work credits. If he is disabled before age 31 he can now qualify for benefits with fewer years of work than before.
- Widows and widowers who are 50 or older can receive benefits based on disability if the spouse was covered under Social Security.
- Persons handicapped continuously since before 18 may get benefits under the "childhood disability" provisions when a parent covered under Social Security dies or becomes entitled to retirement or disability benefits.
- Benefits are payable if the disability has lasted, or is expected to last, 12 months or longer or results in death.
- Benefits generally begin with the seventh full month of disability.
- Benefits for a worker are the same amount that retirement benefits would be if he were 65.
- All applicants are considered for vocational rehabilitation services.

Benefits begin with the seventh full month of disability. (A 6-month waiting period is required by law.) Payments can last as long as the disability continues. At 65 payments to disabled workers are converted into retirement benefits without any change in amount.

Helping Your Patients

Many physicians routinely advise any patient who they think might qualify for disability benefits to consult his Social Security Office. Such advice can be a great service to the patient. Since disabled patients are often having financial difficulties your suggestion that they file may result in their getting urgently needed funds.

The people in any Social Security Office can tell a claimant what the eligibility requirements are and can help him apply for benefits. If a person is unable to come to the office because he is homebound or hospitalized, a Social Security representative can arrange to visit him.

---

* Director, Bureau of Disability Insurance, Social Security Administration, 6401 Security Blvd., Baltimore, Md. 21235.

Up to the present the most frequent causes of disability have been arteriosclerotic and hypertensive heart disease, emphysema, schizophrenic disorders, pulmonary tuberculosis, rheumatoid arthritis, and osteoarthritis. Currently in the District of Columbia about 4,500 disabled persons, plus about 2,300 dependents, receive about $500,000 a month in benefits under the Social Security Disability Program.

Reporting Data

When a patient applies for disability benefits he is asked where he has received treatment. He also signs a consent form that authorizes any physician having medical records pertaining to him to furnish reports for official use in evaluating his disability.

Disability decisions are made by an evaluation team consisting of a physician and a counselor skilled in vocational evaluation. The team works in an agency of the state in which the applicant lives—in the District of Columbia at the D. C. Department of Vocational Rehabilitation.

Typically, the evaluating physician is a private practitioner serving the agency on a part-time basis. He reviews the medical, hospital and laboratory reports and, in conjunction with the vocational evaluator, decides whether the applicant's impairment is disabling under the law. The evaluating physician neither sees nor examines the patient. He depends entirely on the evidence reported by you and others who have examined or treated the applicant.

Experience has shown that the extent of a patient's disability can be determined largely on evidence from his own physician's records. By giving a detailed report of objective data in your files you can help speed the decision on your patient's claim. On the other hand, long delays can result if the physician responsible for evaluating the evidence must write back to you or seek from other sources medical information about symptoms, signs and laboratory findings that you may already have in your files.

For best results report the same information that you would send any colleague to give him a complete medical picture of the patient. This report should contain not only a diagnosis but also a complete history, results of physical examination, and relevant diagnostic tests.

You may wish to save time in preparing the report by enclosing photocopies of pertinent portions of the patient's chart, including laboratory reports, electrocardiograms, X-rays, and the like. Originals are welcome and will be promptly returned if requested.

Under the law, your patient—not the Government—is responsible for providing the initial medical evidence in support of his claim. This is true even though you may receive a request for a report directly from a Social Security office. This is done as a courtesy to the patient at his request to help expedite his claim. Thus, the Government cannot assume responsibility for any charge you may wish to make for furnishing medical information from your records.

Sometimes more medical evidence is needed for an evaluation than is available in the patient's chart. In this situation the physician evaluating the claim may ask you to perform the needed tests and examinations at Government expense, or he may find it necessary to refer the patient for an independent consultative examination, also at Government expense. When the latter is done the consultant's report can be sent to you if you wish.

"Childhood Disability"

In addition to covering disabled workers and disabled widows, the Social Security program has a provision for "childhood disability" benefits—a somewhat anomalous title since these benefits are paid after the person reaches 18. Ordinarily, children cannot get Social Security benefits as dependents after reaching 18 unless they are full-time students, and then only until they reach 22. But under the childhood disability provisions a person continuously disabled since before reaching 18 can be eligible for benefits after he reaches 18.

Childhood disability payments begin when the beneficiary's parent covered under Social Security dies or becomes entitled to retirement or disability benefits. Benefits can continue as long as the "child" lives if he remains disabled. He himself need not have worked under Social Security.

Since 1957 the childhood disability program has awarded monthly benefits to nearly 300,000 persons too handicapped to be self-supporting. More than 65 per cent of beneficiaries under this program suffer from some form of mental deficiency. Other prevalent conditions of beneficiaries under the program include cerebral palsy, schizophrenic disorders, and epilepsy.

Motivating Rehabilitation

In addition to paying cash benefits, one of the main goals of the disability program is to help restore as many applicants as possible to gainful work. At the time your patient's claim is being evaluated he is also being considered for possible services by the D. C. Department of Vocational Rehabilitation. Such services include medical rehabilitation, counseling, teaching of new employment skills, training in the use of prostheses, and job placement.

Rehabilitation services to disability applicants are generally financed from State-Federal appropriations. Additional resources are also provided through Social Security trust funds to pay the costs of rehabilitating certain disability beneficiaries.

As the patient's physician you are in a good position to reinforce his interest in returning to productive work, if this is feasible. Incorporated in the Social Security Disability Program are several incentives for rehabilitation that doctors often cite to help motivate patients. For example, when a worker on the benefit rolls who has not recovered tries to reënter the labor force his benefits will continue for as long as a year even though he is holding a job. Typically, he will go through a 9-month trial work period (not necessarily consecutive months) during which he can test his abilities while still receiving benefits. Then, if he proves capable of working, he will receive benefits for a final 3-month adjustment period. If he has not regained his ability to work, benefits will be continued. The purpose of this year-long continuation of benefits is to overcome the anxiety of beneficiaries who fear they will be cut off from all income if they attempt unsuccessfully to work.

As a further incentive beneficiaries who have recovered or returned to work get special consideration if they again become disabled. If disability recurs within 5 years (7 years for widows), they need not go through the normal 6-month waiting period before benefits resume. This helps reassure persons who fear they will have no means of support if they have a relapse.

HEALTH DEPARTMENT NOTES

Physical Medicine and Rehabilitation

Physical Medicine and Rehabilitation (P.M.R.) is one of 17 treatment divisions at D. C. General Hospital. The work performed by the staff of physiatrists and therapists provides a much needed service to patients who have undergone major surgery or have been injured in serious accidents.

The renovation and construction of the medical and surgical buildings at the Hospital made available new and modern rehabilitation facilities at which an average of 150 patients are treated each day.

In meeting the needs of patients the P.M.R. division renders diagnostic and treatment services to both inpatients and outpatients referred from all services and clinics in the Hospital. The ultimate goal in each case is to return the individual to a more useful life, retraining the patient to perform new job skills or to relearn old ones.

Physiatrists see patients on a consultation basis. In addition to prescribing the type of therapy and supervision needed, some consultations are given in electromyography and nerve conduction and time studies.

In physical therapy registered therapists render prescribed treatment, utilizing the physical agents of heat, light, water, sound, electricity, massage,

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The Executive Board of
The Medical Society of the
District of Columbia

announces the appointment of

A. LOUISE ECKBURG

Executive Editor of the Medical Annals

This action was taken at the meeting of
the Board, July 22, 1968 (see page 502).

therapeutic exercise, and functional activities, such as gait training. They also perform specialized tests which aid physicians in diagnosis and evaluating the prospects for recovery.

Registered occupational therapists utilize a number of skills and activities for therapeutic exercise and psychological support. They teach self-care and homemaking activities and perform prevocational evaluation and training. Special hand splints and self-help devices are developed to aid patients to overcome difficult problems.

The work therapy program is important to those who must relearn old skills due to physical limitations or learn new skills to qualify for employment. Following completion of physical and occupational therapy programs, the patient is ready for work assignments in various locations in the hospital. When this work therapy program nears completion the D. C. Department of Vocational Rehabilitation interviews patients who are interested in seeking employment, and arrangements are made for job interviews with prospective employers.

Although the importance of physical and occupational therapy is an accepted part of a patient's recovery, the growing shortage of physiatrists and therapists is posing a serious problem. At D. C. General Hospital there are some 20 vacancies for the type of specialties noted above. Since the start of the Medicare program many of these highly trained individuals resigned their posts for positions with nursing homes. The lack of teaching facilities in the District of Columbia is a deterrent to providing a resource of trained personnel. There are no approved schools for physical and occupational therapy in Washington, D. C. However, 2 of the 3 local medical schools offer residency programs for those who wish to specialize in physical medicine. There is a growing need for physiatrists. It is hoped that plans can be formulated to provide proper training facilities for these skilled professionals and encourage qualified individuals to utilize them.

Murray Grant, M.D., D.P.H.
D. C. Director of Public Health

NEW MEMBERS

Active

Bernard Grand, M.D. Office: 1711 Rhode Island Avenue, N.W. Born in New York City. Medical degree: New York University, 1962. Specialty: Internal Medicine.

Aubrey L. Jennings, M.D. Office: 5401 Wisconsin Avenue, N.W. Born in Bartlett, Texas. Medical degree: Oklahoma University, 1929. Specialty: Ophthalmology.

Donald R. Payne, M.D. Office: 915 19th Street, N.W. Born in Dallas, Texas. Medical degree: University of Texas Southwestern Medical School, 1962. Specialty: Obstetrics and Gynecology.

Brantley Paul Vitek, M.D. Office: 2520 L Street, N.W. Born in Baltimore, Md. Medical degree: University of Maryland, 1961. Specialty: Orthopedic Surgery.

Ernest Michael Zimmerman, M.D. Office: 1800 Eye Street, N.W. Born in Toronto, Canada. Medical degree: Dniversity of Toronto, 1963. Specialty: Ophthalmology.

Charles Paddack, M.D. Office: U. S. Naval Station, Box 18, F.P.O. New York 09540. Born in Santurce, Puerto Rico. Medical degree: Georgetown University, 1964. Specialty: Psychiatry.

Associate

Ira N. Brecher, M.D. Office: 800 Pershing Drive, Silver Spring, Md. Born in New York City. Medical degree: Howard University, 1963. Specialty: General Surgery.

Arthur Kaufman, M.D. Office: 115 Centerway Road, Greenbelt, Md. Born in Jersey City, N. J. Medical degree: State University of New York Downstate Medical Center, 1963. Specialty: General Practice.

Resident-Intern

James Shannon Butler, M.D. Office: Bassett Army Hospital, Fort Wainwright, Alaska 99782. Born in Woodbury, N. J. Medical degree: George Washington University, 1963. Specialty: Otolaryngology.

Ralph Lew, M.D. Office: Washington Hospital Center. Born in Oakland, Calif. Medical degree: George Washington University, 1966. Specialty: General Practice.

George Stuart Scott, M.D. Office: National Institutes of Health, Bethesda, Md. Born in Augusta, Ga. Medical degree: Duke University, 1966. Specialty: Internal Medicine.

News & Personals

Summary of Executive Board Actions

Proceedings of July 22 Meeting

Dinner for the Board members was served in the Exhibit Hall of the Medical Society. Dr. Edward W. Nicklas, Chairman, presided at the meeting which was attended by Drs. William S. McCune, Frank S. Bacon, Jed W. Pearson, Jr., Gudrun Augustin-Rueckert, Paul R. Wilner, Henry D. Ecker, Raymond T. Holden, John J. Hughes, and Mr. Thomas Sadler, Secretary. The meeting was called to order by Dr. Nicklas at 7:30 p.m.

The minutes of the June 24, 1968 meeting were approved as submitted.

Committee Meetings: The Secretary reported the following committees had met since the last meeting of the Board:

June 24	Recruitment Subcommittee, Regional Medical Program
25	Committee on Aviation Medicine
26	Committee on Liaison with the Clergy
26	Public Welfare Committee
28	Recruitment Subcommittee, RMP
July 1	Recruitment Subcommittee, RMP
9	Board of Credentials
9	Committee on Aviation Medicine
10	Funeral Committee
10	Fee Review Committee
11	Traffic Committee
16	Grievance Committee
18	Medical Bureau Committee

Board of Credentials: On recommendation of the Board of Credentials, 6 applicants were elected to Active membership, 2 to Associate membership, and 3 to Resident-Intern membership. Their names appear on page 500. Approval was given to the following changes in membership status:

Transfers

Active to Associate: Donald Gerald Levitt
Associate to Active: Leonardo C. Maguigad

Resignations

Active: Vernon Henry Balster
Associate: Bernard H. Zeavin
Resident-Intern: Faruk S. Abuzzahab

Report on AMA Convention: Dr. Holden read to the Board the delegates' report on the June AMA Convention in San Francisco (see page 491). After general discussion on the report, the Chairman requested further information on AMA recommendations for submission at the next meeting. Dr. Hughes agreed to present one recommendation per meeting.

President McCune read the following letter from Dr. Holden:

> "Dear Bill:
>
> "This letter is one I write with mixed emotions. Having been associated with the affairs of the Society in one capacity or another for so many years, it will seem strange to no longer be intimately a part of it. But, I must now submit, regretfully, my resignation as delegate from our Society to the House of Delegates of the American Medical Association.
>
> "It is a matter of pride to know that as a member of the Medical Society of the District of Columbia I shall be representing medicine at a national level, and I assure you that I will do my best to fulfil the confidence which you and my colleagues have placed in me.
>
> "Clifton Gruver, my up-to-now alternate, is acquainted with the procedures of the House of Delegates as he has attended several sessions, and I am sure that he can represent the Society well. Of course, John Hughes was really indoctrinated during his first session and will be a fine representative.
>
> "In closing I should like to take the opportunity to thank you, the Executive Board, Mr. Sadler, and the members of the Society collectively and individually for their efforts on my behalf.
>
> "With the best personal regards, I remain,
>
> Sincerely,
> s/Ray"

AMA Delegate to Replace Dr. Holden: President McCune read a letter addressed to him from Dr. Clifton R. Gruver in which he gave several personal reasons for resigning as alternate delegate to the AMA. The Executive Board accepted Dr. Gruver's resignation with regret and requested that the President send Dr. Gruver a letter of commendation for his contributions and services to the Society.

A motion was made and carried unanimously to appoint Dr. C. Willard Camalier, Jr. to serve as delegate to the AMA for the unexpired term of Dr. Holden. A motion was also made and unanimously carried that Dr. William H. Cooper be appointed to serve as alternate delegate to the AMA for the unexpired term of Dr. Gruver.

DOCPAC Dues Statements: A motion was made and carried that DOCPAC membership dues be included on Society dues statements as a separate item. It should be indicated on the bill-head that DOCPAC dues are not tax-deductible but are contributory donations and that membership in DOCPAC is not obligatory.

Membership Committee: On the recommendation of the Membership Committee the Board voted to publish periodically in the MEDICAL ANNALS a list of Active members of the Society (with their address, telephone, and specialty) at the discretion of the Finance Committee.

The Board requested Mr. Sadler to explore the "Orange Book" to determine whether membership in the Medical Society can be indicated for physicians listed therein and whether breakdown by locale is possible.

The Board rejected the recommendation of the Membership Committee that a new membership category of "Academic" be established for members who spend 50 per cent or more of their time in research.

Dr. McCune was requested to discuss this item further with Dr. Irving Burka, Chairman of the Membership Committee. It was also suggested that the Membership Committee make a more vigorous effort to recruit more members.

The Board voted to table the recommendation that a 25 per cent remission of dues be credited to all members who are full-time employees of Federal health services until the total number of members in such a category is ascertained.

Medicaid Fee Schedule: For informational purposes Dr. Bacon read his memorandum of July 11, 1968 regarding Title XIX and elaborated on its contents. The Board authorized the President to notify Society members that the Medicaid program has been approved and to participate when requested to do so.

A motion was made and carried that the Medico-Chirurgical Society of the District of Columbia be asked if it would like to purchase, at cost, copies of the Relative Value Scale of the Medical Society of the District of Columbia for use in the Medicaid program.

Change in Title for Assistant Editor: On Mr. Sadler's recommendation, and with the concurrence of Dr. Wallace M. Yater, Editor, it was voted to change the title of Miss Louise Eckburg from Assistant Editor to Executive Editor of the MEDICAL ANNALS.

Referral Policy for Persons Seeking Physician for Examination, Treatment, or Follow-up of Malignant Disease: Discussion was held in regard to a request, with supporting proposed questionnaire, from the D. C. Cancer Society for establishment of a referral roster composed of physicians whose special interest or subspecialty is the diagnosis and treatment of cancer. It was decided that Dr. Pearson discuss this matter fully with Dr. C. Roger Kurtz, President of the Cancer Society, and to release the questionnaire when Dr. Pearson is thoroughly satisfied with its contents.

1968 Davidson Lecture: Considerable discussion was held regarding a request from Dr. Elizabeth S. Kahler that income from the Davidson Fund be used for honoraria alone and that the Society continue underwriting all other expenses for the Davidson Lecture. A review was presented of the total expenses incurred for sponsoring the Davidson Lectures from 1961 to 1967. The Board voted that expenditures for the Davidson Lecture this year be limited to income received as interest on monies in the fund, and that the previous action of the Executive Board be reaffirmed.

Nominations for Vacancies on AMA Councils and Committees: It was agreed that the Society consider submitting names of nominees to fill positions on AMA councils and committees, and it was further agreed that the President select the nominees after conferring with appropriate committee chairmen.

Should the Society Approve the Use of Interns/Residents on the Mobile Coronary Care Units? There was considerable discussion as to the wisdom of the Society's approving the use of interns and residents on the mobile coronary care units. It was voted that the Medical Society approve in principle the use of interns and residents on the mobile coronary care units, but the Society is concerned about the legal protection of these interns and residents and would like to be advised in writing that they will be covered legally in every way by their hospital.

Dr. McCune was requested to send appropriate hospital administrators a letter embodying the above motion.

Preliminary Report on Establishing Private Physicians and Admitting Private Patients to D. C. General Hospital: Dr. McCune summarized the contents of a June 10, 1968 memorandum from Dr. John Nasou on establishing private physicians and admitting private patients to D. C. General Hospital. A motion was carried that, in the opinion of the Board, before instituting the program described D. C. General should first relieve other area hospitals of their overburden of patients.

Lobby Displays: The Board approved, without vote, Mr. Sadler's recommendation that the Society schedule appropriate exhibit displays in the lobby of the Society's building.

Candidacy of Dr. Bornemeier for AMA President-elect: It was decided to leave to the good judgment of the delegates their support of Dr. Bornemeier's candidacy for President-elect of the AMA.

Zoning Restrictions and Occupancy Law: Dr. McCune reviewed with the Board information obtained from a recent questionnaire to the membership in regard to zoning restrictions and the occupancy law. Dr. McCune also reported that Dr. Samuel Diener had been appointed by Dr. Harvey Ammerman to investigate problems created by this archaic law. Dr. Ammerman himself will, upon return from vacation, work with Dr. Diener on this matter.

Proposed Establishment of an Area Uniformed Services Medical Academy: The Board discussed briefly Dr. Louis M. Rousselot's letter of July 8 on this subject but no specific recommendations or conclusions were made. It was mentioned, however, that possibly the greatest deterrent would be lack of sufficient clinical material in Washington to support a fourth medical school or academy. The Board requested Dr. McCune to acknowledge receipt of Dr. Rousselot's letter.

Confirmation was given to the following actions taken by telephone canvass:

1. Mr. Thomas Sadler was appointed Managing Editor of the Medical Annals.

2. Dr. Arthur E. Wentz was appointed Coordinator for the Regional Medical Program, succeeding Dr. Thomas W. Mattingly, resigned.

3. The 1969 Scientific Assembly will be held at the Mayflower Hotel, September 24, 25 and 26, 1969.

Other Business

1. The Board approved the sale of the Society's commemorative vases at the forthcoming Scientific Assembly by 2 employees attired in the styles of 1817 and 1968.

2. The Board agreed to establish the following procedures for awarding plaques to Honorary members: Plaques will be presented yearly at the Scientific Assembly to those members who have been elected to Honorary membership. A letter from the President will inform such member of his Honorary membership status and invite him to be present to receive his plaque at the ASA luncheon.

3. A motion was made and carried to support the request of Mr. George McGovern for the establishment of an area-wide Fee Review Committee.

4. It was voted to refer to the Medical Bureau the request from the Medical Council for comments, suggestions, and recommendations regarding Yellow Page listings in the Telephone Directory.

5. Mr. Sadler reported receipt of a check for $1,000 bequeathed to the Medical Society under the terms of the will of the late Dr. Frank M. McChesney. He noted further that Dr. McChesney had donated $500 in 1957 and an additional $500 in 1959. At the Chairman's request Mr. Sadler read to the Board the contents of a letter addressed to former Secretary Wiprud which accompanied Dr. McChesney's original donation:

> "The designation in one's will of favorite projects is admirable. However, I feel that as the Lord prospers me I would like to share from time to time in worthy causes. Inclosed find a check. I will leave it to your judgment as to where it can best be used."

6. The Board agreed that this year's Annual Business Meeting be held on a Tuesday night, beginning at 5:30 p.m., in an effort to draw greater attendance.

7. Mr. Sadler reported that Lou Brott (public relations) would like to know the Society's interest in coöperating in a WTOP radio show on abortion, with participants to be very firm negatively or positively on the subject. The Board voted to ask Dr. Zigmond M. Lebensohn to present the positive side, if he is interested in doing so, and to suggest someone for the negative side.

8. The Board approved in essence the proposal from UGF that solicitation this year be centralized through the Medical Society with a letter to the membership regarding UGF signed by the

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Society's President. Mr. Sadler pointed out that UGF would help to prepare such a letter and would pay for the postage, return envelopes, and handling. The Society would merely process the envelopes through its mailing list of Active members.

There being no further business, the meeting was adjourned at 11:35 p.m.

MEETINGS

The 28th Annual Meeting of the American Medical Writers Association will be held, September 19–22, at the Mayflower Hotel in Washington. The theme for the session will be "National Perspectives in Medical Communications."

Dr. Herbert L. Ley, Jr., newly appointed Commissioner of the Food and Drug Administration, will participate in a panel on "Doctors, Drugs and Government: Is There a Happy Ending?"

Other prominent speakers will be Dr. Louis Rousselot, Deputy Assistant Secretary for Health and Medical Affairs in the Department of Defense, and Dr. Philip R. Lee, Assistant Secretary for Health and Scientific Affairs, Department of Health, Education, and Welfare.

"What the Press Wants to Know and Why" will be the subject of another panel, with Mr. Byron Scott, of Chicago, medical news editor of J.A.M.A., as moderator. Panelists from Washington will be Colonel Hamilton B. Webb, USAF, MC; Mr. J. Stewart Hunter, Office of Public Information, HEW; Mr. Jonathan Spivak, of the *Wall Street Journal*; Mr. John Walsh, news editor, *Science Magazine*; and Mr. Wallace Werble, editor and publisher, *Drug Research Reports*.

Members of the Medical Society are cordially invited to attend the meeting. There is a registration fee of $10.00 for members and $30.00 for non-members, $20.00 of which may be applied to first-year membership dues.

The American Medical Association has scheduled 3 meetings of special interest early in October. The first, **the 28th Annual Congress on Occupational Health,** is a 2-day meeting, beginning September 30 and extending through October 1. It will be held at the Waldorf-Astoria Hotel in New York City. There is no registration fee.

Dr. R. Lomax Wells, of Washington, Chairman of the AMA Council on Occupational Health, will preside at the opening session on Monday morning. The first session will be addressed by Dr. Dwight L. Wilbur, President of the AMA.

Dr. Wells will also preside at the Physician's Award Ceremony, Tuesday, at 11:30 a.m., when the Annual Physician's Award of the President's Committee on Employment of the Handicapped will be presented. Dr. Howard A. Rusk, Director of Rehabilitation at New York University Medical Center, will make the presentation. He will also be the principal speaker at the luncheon to follow; his subject will be "Rehabilitation in Vietnam."

On October 2 and 3, **the Fourth National Congress on Health Quackery** will be held at the Drake Hotel in Chicago. "Protecting the Consumer" will be the theme of the meeting. Attendance will be by invitation. Inquiries may be addressed to Dr. Joseph A. Sabatier, Jr., Chairman, Committee on Quackery, AMA, 535 North Dearborn Street, Chicago, Ill. 60610.

The Second National Congress on Medical Ethics, which will also be held at the Drake Hotel, will meet October 5 and 6. The program will be a practical consideration and application of ethical principles in everyday practice. It will run the gamut of questions from relationships with ancillary personnel to the ethics of transplanting hearts and lungs. Distinguished speakers from all over the country have agreed to participate in the program. Attendance will be limited and early registration is encouraged. Write to Dr. E. D. Shelley, Chairman, Judical Council of the AMA.

All interested physicians are urged to attend the **Tenth International Congress on Diseases of the Chest of the American College of Chest Physicians,** to be held, October 4–8, at the Washington Hilton Hotel.

Dr. Raman Viswanathan, Professor and Emeritus Scientist, Vallabhbhai Patel Chest Institute, University of Delhi, India, will present the Third Founders' Lecture at the inaugural session of the Congress on Friday evening, October 4.

Premiere showing of the motion picture, "Transplantation of the Human Heart," is scheduled at a special session. Members of the distinguished heart transplantation team, Drs. Denton A. Cooley, Robert D. Bloodwell, Grady L. Hallman, and James J. Nora, affiliated with the Texas Heart Institute of St. Luke's Hospital, Texas Children's Hospital, and Baylor University College of Medicine, Houston, will discuss their experiences with several human heart transplants.

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Dr. Roy G. Klepser, of Washington, is General Chairman for the Congress. He is being assisted by the following committee chairmen: Lt. General Richard L. Bohannon, USAF, MC, Rear Admiral Robert Canada, MC, USN, and Drs. Gerrit W. H. Schepers, Benjamin Manchester, Joseph W. Peabody, Jr., W. LeRoy Dunn, William S. Lyons, Edward Dunner, William Byrne, and George W. Ware.

Registration forms may be obtained from the American College of Chest Physicians, 112 East Chestnut Street, Chicago, Ill. 60611. Fees for members and non-member physicians are $45.00 and $55.00, respectively.

The American College of Physicians will sponsor a regional scientific meeting for specialists in internal medicine from Maryland and the District of Columbia, October 12, at Georgetown University School of Medicine.

Dr. H. Marvin Pollard, of Ann Arbor, Michigan, President of ACP, will be one of the special guests. Dr. Pollard is Professor of Internal Medicine at the University of Michigan School of Medicine and head of the Section of Gastroenterology at the University of Michigan Medical Center.

In general charge of the meeting are Dr. Thomas W. Mattingly, of Washington, and Martin L. Singewald, of Baltimore.

The Johns Hopkins University School of Medicine and the Maryland Chapter of the American Academy of Pediatrics will co-sponsor a postgraduate course on "Difficult Problems in Pediatrics and Pediatric Surgery," October 10–12, at the University.

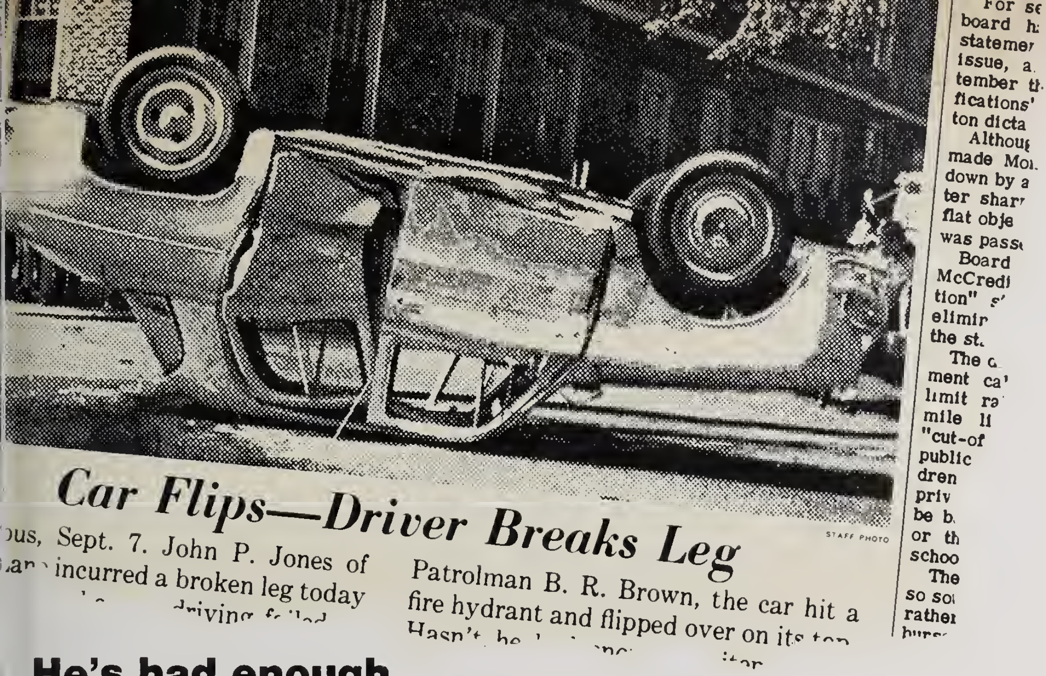
Subjects to be discussed will include management of respiratory distress, use of antibiotics in children's surgery, diagnosis and management of eye muscle problems, and special problems in the surgery of newborn infants.

Additional information concerning this program may be obtained from Dr. J. Alex Haller, Jr., Professor of Pediatric Surgery, Johns Hopkins Hospital, Baltimore, Md. 21205.

The American Society of Anesthesiologists will meet in Washington, D. C., for its **1968 Annual Meeting** at the Washington Hilton Hotel, October 21, 22 and 23. Papers will be scheduled by category instead of the usual "Work in Progress" and "Work Completed." Two guest speakers will be Dr. Douglas R. Waud, of the Department of Pharmacology, Harvard Medical School, and Dr. Arnold M. Weissler, Director of Cardiology in the Department of Medicine at Ohio State University. Seventy-six papers have been selected from those submitted in competition. This year each paper will be allotted 10 minutes, with 4 minutes for discussion.

Dr. Seymour Alpert, Chairman of the Local Arrangements Committee, promises that the meeting will be highlighted by some unusual events. A special concert has been arranged for Monday, October 21, by the National Gallery of Art Symphony Orchestra, and there will be a private viewing for members and guests of the world-famous collection at the National Gallery of Art.

A lecture on "The History of Anesthesia" will be given at the National Library of Medicine, in Bethesda, Wednesday afternoon, October 23. There will also be guided tours of the Library. The Arrangements Committee is planning other tours of historic interest and many social events.



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Car Flips—Driver Breaks Leg

STAFF PHOTO

ous, Sept. 7. John P. Jones of
incurred a broken leg today

Patrolman B. R. Brown, the car hit a
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He's had enough excitement for one day.

For the patient who has been through an accident, the worry and anxiety following the experience may actually heighten the perception of pain. This is why there's a classic 1/4 grain *sedative* dose of phenobarbital in Phenaphen with Codeine—*to take the nervous "edge" off,* so the rest of the formula can control the pain more effectively.

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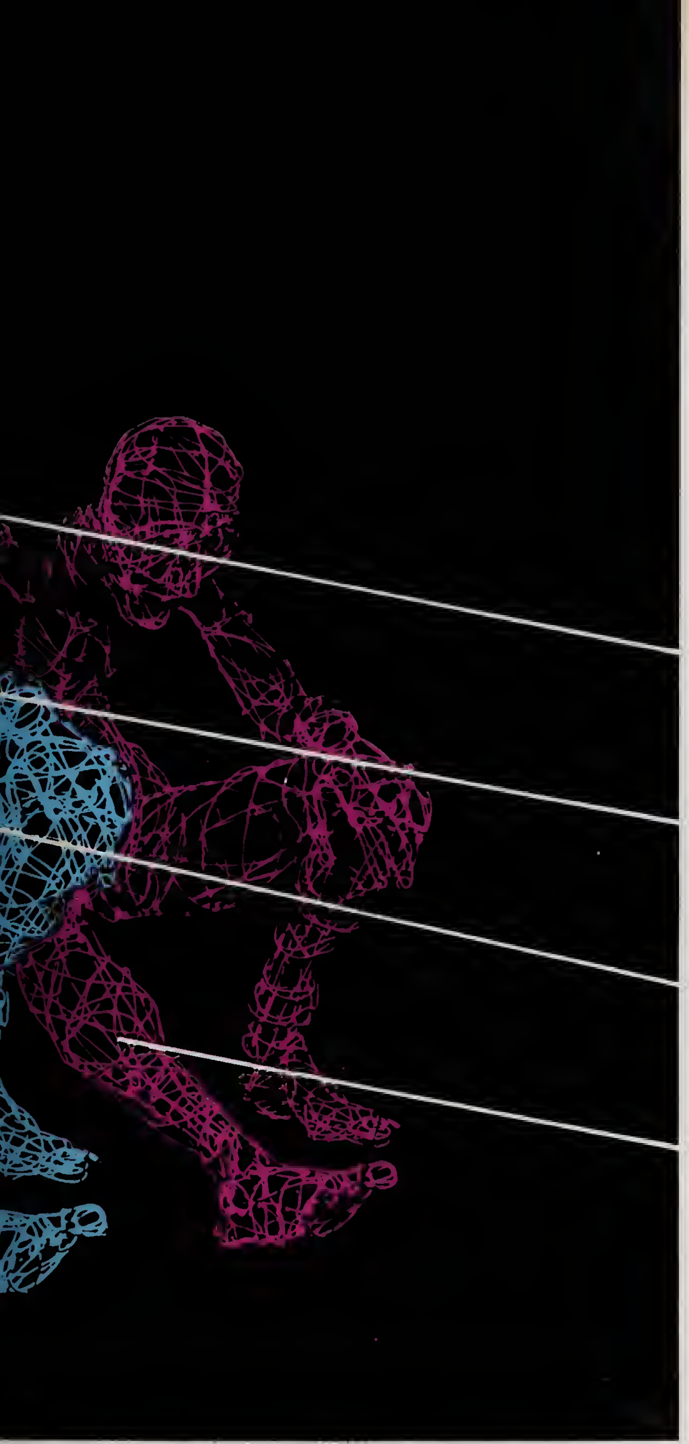

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GENERAL NEWS

The National Kidney Foundation extends its annual invitation to qualified medical investigators and institutions to apply for research grants in kidney diseases. The deadline for submission of applications is November 15, and awards will be made effective July 1, 1969. Last year Georgetown University Medical Center received $18,000 in Kidney Foundation research and fellowship grants. Applications may be obtained from the Foundation at 1145 19th Street, N.W., Washington, D. C. 20036.

Georgetown University School of Medicine has been awarded a $300,000 grant over a 3-year period from the National Institute of Mental Health for a study of pre-trial diversion of mentally ill offenders. The project is designed to carry out recommendations of the President's Commission on Law Enforcement and the Administration of Justice. Dr. James L. Foy, Associate Professor of Psychiatry, will be assisted in the project by Mr. Edward De Grazia, a former professor at the Georgetown University Law Center who is now associated with the new Federal City College.

George Washington University School of Medicine is one of 30 U. S. medical schools to share in a $10 million grant from the Richard King Mellon Charitable Trusts, a Pittsburgh foundation, for expanding and strengthening medical teaching. Ten of the schools, including GW, will receive $500,000, awarded in $100,000 annual grants over the next 5 years. The grants may be allocated at the discretion of the deans of the medical schools with the hope that they will attract promising young physicians to the faculty as full-time teachers in medicine.

Dr. Erich G. Krueger, nationally known neurosurgeon, has been named director of the spinal cord injury program of the Veterans Administration. Dr. Krueger has been serving as Chief of Neurosurgery at the VA hospital in Bronx, N. Y., and as Clinical Professor of Neurological Surgery of the College of Physicians and Surgeons of Columbia University.

Dr. Krueger succeeds Dr. Emanuele Mannarino in this position. Dr. Mannarino, an Associate member of the D. C. Medical Society, will remain at VA as Chief of the Neurosurgery Section.

Dr. Charles A. Hufnagel assumed office as **President of the Washington Heart Association** at its annual meeting, June 27, in the Medical Society's Exhibit Hall. Dr. Hufnagel, internationally known for his pioneer work in heart surgery, is Professor of Surgery at Georgetown University School of Medicine and Director of the Surgical Research Laboratory at the Hospital.

Dr. Hufnagel, who took office in June as President of Heart Association

Other officers elected were: Dr. George E. Schreiner, President-elect; Dr. John B. Johnson, Vice President; Mr. William T. Hannan, Secretary; and Mr. John E. Sumter, Jr., Treasurer.

Dr. Clayton B. Ethridge, who retired recently as Associate Dean of George Washington University School of Medicine and Medical Director of the University Hospital, was presented an engraved plaque for his 20 years' "service with distinction" to the Washington Heart Association.

The Women's Medical Society of the District of Columbia has elected the following officers for 1968–69:

Dr. Catharine Knop, *President*; Dr. Agnes E. Schweitzer, *Vice President*; Dr. Francis S. Norris, *Secretary*; and Dr. Theresa T. Woo, *Treasurer*.

Dr. Knop succeeds Dr. Petrena Abbe Shea as President of the Society.

Colonel Loren F. Parmley, Jr., MC, USA, Chief of the Department of Medicine, Walter Reed General Hospital, was recently awarded the Legion of Merit in ceremonies held at the Hospital. He is a nationally recognized specialist in internal medicine and cardiology and has often been called upon to supervise and take part in the care and treatment of prominent leaders of the United States and foreign governments. A Texan, he received his medical degree from the University of Virginia in 1943.

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Warnings: Acute anaphylaxis (may prove fatal unless promptly controlled) is rare but more frequent in patients with previous penicillin sensitivity, bronchial asthma or other allergies. Resuscitative (epinephrine, aminophylline, pressor amines) and supportive (antihistamines, methylprednisolone sodium succinate) drugs should be readily available. Other rare hypersensitivity reactions include nephropathy, hemolytic anemia, leucopenia and thrombocytopenia. In suspected hypersensitivity, evaluation of renal and hematopoietic systems is recommended.
Precautions: In suspected staphylococcal infections, perform proper laboratory studies including sensitivity tests. If overgrowth of nonsusceptible organisms occurs (constant observation is essential), discontinue penicillin and take appropriate measures. Whenever allergic reactions occur, withdraw penicillin unless condition being treated is considered life threatening and amenable only to penicillin. Penicillin may delay or prevent appearance of primary syphilitic lesions. Gonorrhea patients suspected of concurrent syphilis should be tested serologically for at least 3 months. When lesions of primary syphilis are suspected, dark-field examination should precede use of penicillin. Treat beta-hemolytic streptococcal infections with full therapeutic dosage for at least 10 days to prevent rheumatic fever or glomerulonephritis. In staphylococcal infections, perform surgery as indicated.
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Colonel Parmley, who is a Consulting Editor to Medical Annals, representing Walter Reed General Hospital, retired from active military service on July 31.

The Dean of Georgetown University School of Medicine has announced the **appointment of Dr. Robert Clayton as Director of the Child Development Center** for mentally retarded children. The Center, now under construction at Georgetown, adjacent to the Hospital, is affiliated with American, Catholic, George Washington, and Howard Universities, and Gallaudet College, all in Washington. Plans call for the Center to be completed by the spring of 1969 at a cost of $2.7 million, of which $1.5 million has been provided by the Federal Government.

Dr. Clayton is Assistant Professor of Pediatrics at Georgetown. He is regional medical consultant to Project Head Start, of the Office of Economic Opportunity, and active in several groups concerned with mental retardation, child health, and blindness.

PERSONALS

Dr. Maurice Mensh, a member of the D. C. Board of Police and Fire Surgeons since 1946 and Vice Chairman of the Board for the past 6 years, has been appointed Chairman of the Board by Mayor Walter E. Washington. He succeeds Dr. Benjamin F. Dean, Jr.

Appointed Vice Chairman of the Board was **Dr. Joseph F. Dyer, Jr.,** Clinical Assistant Professor of Medicine at Howard University School of Medicine.

Dr. Robert E. Pumphrey, of Children's Hospital, flew to Alaska with a team of otologists and anesthesiologists at the end of June to spend his vacation treating children for deafness. He made his headquarters at the U. S. Public Health Service Hospital in Kotzebue, about 150 miles north of Nome.

Accompanying Dr. Pumphrey were Dr. David Beall, of the National Institutes of Health; **Dr. Walter R. Helmig,** of Sibley Hospital; **Dr. James J. McFarland, Jr.,** of George Washington University Hospital; **Dr. Kamel J. Hassan,** of Children's Hospital; and Dr. Charles Tschopp, of the Public Health Service. The trip was sponsored by the Public Health Service's Division of Indian Health. All of these physicians, except Dr. Beall and Dr. Tschopp, are Active members of the D. C. Medical Society.

Dr. William P. Herbst, Jr., received the highest civilian award of the Army, "The Outstanding Civilian Service Medal and Citation," June 15, at a dinner at the Washington Hospital Center. According to the citation, Dr. Herbst, Consultant in Urology to Walter Reed General Hospital, "has distinguished himself by exceptionally meritorious performance of outstanding service from 1928 to 1968." The Center and Walter Reed Hospital have a joint residency training program in urology.

At a luncheon at the Naval Officers' Club in Bethesda, July 2, Dr. Herbst was awarded the "Certificate of Merit" by the Bureau of Medicine and Surgery of the U. S. Navy "for loyal and dedicated service as consultant and lecturer in urology at the Naval Hospital, National Naval Medical Center, Bethesda, Md." from 1942 to 1968. Dr. Herbst will continue as "Professor Emeritus" of Urology at the Naval Hospital.

Senator Lister Hill, of Alabama, who is retiring after more than 40 years of public service, was the guest of honor when the Board of Regents and staff members of the National Library of Medicine assembled to pay tribute to him on June 20. Among the distinguished guests were Senator John Sparkman, Junior Senator from Alabama; Mr. Wilbur J. Cohen, Secretary of the U. S. Department of Health, Education, and Welfare; and Dr. Worth B. Daniels, first Chairman of the Presidentially appointed Board of Regents of the National Library of Medicine and also Mr. Hill's personal physician. Colleagues from both Houses of Congress, officials of HEW, and many other friends from the biomedical community attended the event.

Senator Sparkman proposed that the Library's planned new annex be designated the Lister Hill Center for Biomedical Communications in order "to perpetuate the name of the man who has done so much for the health of the Nation, who has exhibited an abiding concern for and interest in libraries in general and NLM in particular."

Dr. William Bean, speaking on behalf of the Board of Regents, presented Senator Hill with a hand-lettered scroll citing his legislative accomplishments and paying him homage.

Senator Hill is an Honorary member of the D. C. Medical Society.

Dr. Robert H. Detwiler, first pediatrician in Arlington County, Virginia, where he has practiced since 1936, was honored by 300 of his former patients and their parents at a testimonial dinner for him, July 14. He retired from active practice on June 1.

During his years in Arlington Dr. Detwiler was active in civic, community, and public service affairs, at one time serving on the County's governing body.

A native of Minnesota, he received his medical degree from Georgetown University School of Medicine. He was the first recipient of the Arlington County Medical Society's Welburn Award in 1956. He received the Legion of Honor Plaque of the Arlington Kiwanis Club in 1962 and the Georgetown University Bicentennial Medal in 1956.

Dr. Detwiler joined the staff of the Food and Drug Administration as a medical officer in the Office of Medical Review on June 3. He is an Associate member of the D. C. Medical Society.

Dr. Catharine Knop, of the Yater Clinic, was one of 50 pediatricians from the United States who flew to Europe on a visit to Moscow, Leningrad, Budapest, and Vienna. In these cities they visited and exchanged views with hospital department heads, attended hospital rounds, and joined in hospital staff discussions.

Dr. Knop attended the meeting of the National Conference of Commissions on the Status of Women, June 20–22, in Washington, D. C., representing the Women's Medical Society of the District of Columbia. She is the newly installed President of the Women's Medical Society.

Dr. Bernard Notes, who has practiced obstetrics and gynecology in Washington for 40 years, retired from private practice at the end of June. On July 1 he joined the U. S. Department of Labor as District Medical Director of the Bureau of Employment Compensation.

Dr. Lawrence S. Lilienfield, Chairman of the Department of Physiology and Biophysics at Georgetown University School of Medicine, has just returned from a sabbatical year in Israel where he worked out a student plan with the Tel Aviv faculty. The new program, which has the support of the Association of American Medical Colleges, permits U. S. students to work for 3 months with the new medical school in Tel Aviv. Dr. Lilienfield also initiated research projects, trained students in research techniques, and lectured during his year abroad.

OBITUARIES

Ronald Atmore Cox, M.D.

(1896–1968)

Dr. Ronald Atmore Cox, Washington eye surgeon and teacher, died, July 16, 1968, after a short illness, at the George Washington University Hospital. Dr. Cox lived at 4408 Edmunds Street, N.W., and maintained offices at 919 18th Street, N.W.

Born in Chester, Pennsylvania, August 15, 1896, Dr. Cox spent his early years in Baltimore, Maryland. He was graduated from Baltimore City College in 1915. He attended the Naval Academy and saw active duty during World War I, with the rank of Lieutenant (j.g.). He received his baccalaureate degree from Johns Hopkins University, attended the University of Virginia, and graduated in medicine from George Washington University in 1926.

Dr. Cox practiced ophthalmology in the District of Columbia for 33 years. He was Associate Professor of Ophthalmology at George Washington University from 1935 to 1945, Professor of Ophthalmology from 1950 to 1963, and, for the last 5 years, Professor Emeritus. He was consultant in ophthalmology to the National Institutes of Health, Seaboard Air Line Railroad, Pennsylvania Railroad, and the Baltimore and Ohio Railroad.

In 1955 Dr. Cox traveled to India, where for a month he performed hundreds of eye operations on the indigent hill natives to relieve those blinded by cataracts. In 1966 he was guest speaker at the Tottori University School of Medicine in Yonago City, Japan.

He was a diplomate of the American Board of Ophthalmology, a Fellow of the American College of Surgeons, and a member of the American Medical Association, the American Academy of Ophthalmology, the George Washington University Medical Alumni Association, the Washington Ophthalmological Society, and the Wills Hospital Society of Philadelphia. He had been an Active member of the Medical Society of the District of Columbia since 1935.

Dr. Cox published numerous articles in the field of ophthalmology and addressed many medical meetings throughout the United States. He was an authority on external eye diseases and a substantial contributor to the field of industrial ophthalmology.

He is survived by his wife, Mrs. Ruth M. Cox, of the home address, and a daughter, Mrs. Kenneth Wells Parkinson, of Westmoreland Hills, Maryland.

Kenneth Wells Parkinson

Francis Charles Allman, M.D.

(1909–1968)

Dr. Francis C. Allman, Chief of Radiology at Casualty Hospital, died, June 16, 1968 at the Hospital after a long illness. He lived at 2133 32nd Place, S.E.

Dr. Allman, a native Washingtonian, was born, March 25, 1909. He graduated from Gonzaga High School and received 2 degrees from Georgetown University, Bachelor of Science in medicine in 1932 and Doctor of Medicine in 1934. The following year he interned at Providence Hospital.

He joined the staff of Casualty Hospital as a full-time radiologist after serving in the U. S. Naval Medical Reserve Corps during World War II. He later became Head of the Department of Radiology.

Dr. Allman was a diplomate of the American Board of Radiology and a Fellow of the American College of Radiology. He was a member of the American Medical Association, an Active member of the Medical Society of the District of Columbia from 1937, and a member of the Radiological Society of North America and the Georgetown Clinical Society. He was a member of St. Francis Xavier Church.

He leaves his wife, Mrs. Mary Wahler Allman; 2 sons, Michael J. Allman, of Bowie, Md., and Timothy D. Allman, of Washington; and 5 daughters, Sister Mary St. Francis de Sales, R.G.S., of Philadelphia, Mrs. Kathleen Hutchinson, of Greensburg, Pa., Mrs. Teresa Cuddy of Rockville, Md., and Catherine and Cecilia Allman, both of the home address.

Abraham Ruchwager, M.D.

(1908–1968)

Dr. Abraham Ruchwager, who had practiced psychiatry in the District of Columbia and area since 1954, died, July 8, 1968 at his home, 7 Mel-Mara Drive, Oxon Hill, Maryland.

Dr. Ruchwager was born in Odessa, Russia, September 1, 1908. He received his medical degree from the Royal University of Milan, Italy, in 1932, and he interned at Belgrade, Yugoslavia, in 1933-34. From 1934 to 1939 he was enrolled for postgraduate study in psychiatry at Zagreb, Yugoslavia.

After he came to the United States Dr. Ruchwager continued his training in psychiatry at Crownsville State Hospital, Maryland, from 1947 to 1950, and remained on the hospital staff until 1955. For 10 years, beginning in 1955, he was a medical officer at St. Elizabeths Hospital. During this time he also conducted a part-time practice in psychiatry at 2025 Eye Street, N.W., until he retired in 1966.

During World War II Dr. Ruchwager served as medical officer (Captain) in Yugoslavia and Italy with the Royal Yugoslavian and British Medical Corps.

He was elected an Active member of the Medical Society of the District of Columbia in 1956. He was a member of the American Medical Association and the Jacobi Medical Society of Washington.

He leaves his wife, Mrs. Zdenka Ruchwager; a son, Gary; and a daughter, Mrs. Brian Lurie, of Cincinnati. A brother, Dr. Menasche Ruchwager, and 2 sisters, Mrs. Zina Pesah and Mrs. Rose Zwiagin, live in Tel Aviv, Israel.

Frederick J. Cullen, M.D.

(1888–1968)

Dr. Frederick J. Cullen, an Associate member of the Medical Society of the District of Columbia, died, June 10, 1968 at the Washington Hospital Center.

Dr. Cullen was born in Kokomo, Indiana, December 25, 1888. He graduated from the Winona College of Pharmacy in Indianapolis in 1908. In 1913 he received his medical degree from the University of Colorado, following which he interned for a year at the Northern Pacific Railroad Hospital in Takoma, Washington.

He enlisted for service with the Army Medical Corps in 1916 and served in Mexico and in the Mexican Border campaigns. During World War I he served in France as regimental surgeon and took part in the battles of Aisne, Marne, St. Mihiel, and the Argonne Forest. In 1919 he retired from service because of injuries, with the permanent rank of Major. He received a number of decorations and awards.

After he left the Army he practiced in the State of Washington. He joined the U. S. Public Health Service at Seattle in 1928. The following year he became a staff member of the Food and Drug Administration in Washington and served as Chief of the Drug Control Division until 1934.

From 1934 to 1956 he was Vice President and Medical Director of the Proprietary Association at 1700 Pennsylvania Avenue, N.W. After retiring he continued as medical consultant to the organization.

Dr. Cullen became an Associate member of the District of Columbia Medical Society in 1932. He was a member of the American Association for the Advancement of Science and the Association of Military Surgeons of the United States. He belonged to the Army Navy Country Club, the National Press Club, the Congressional Country Club, and other civic and veterans' organizations.

He is survived by his wife, Mrs. Marie Stone Cullen, of 2914 Garfield Street, N.W.

John J. Blasko, M.D.

(1912–1968)

Dr. John J. Blasko, until recently Director of Psychiatry, Neurology, and Psychology Services at Veterans Administration Hospital, died, July 2, 1968 at the VA Hospital. He lived at 2005 Beechwood Road, Lewisdale, Maryland.

Dr. Blasko was born in Carbondale, Pa., June 29, 1912. After premedical study at Scranton University in Scranton, Pa., he enrolled at St. Louis University School of Medicine, receiving his medical degree in 1935. He interned the following year at Christian Hospital, St. Louis.

After a few years of general practice in St. Louis, he trained in psychiatry at the State Hospital in Fulton, Missouri, from 1939 to 1942, and continued at the State Hospital as Clinical Director until 1945. He served in the U.S. Navy, with the rank of Lieutenant (s.g.), from 1945 to 1946, in charge of psychiatric service at Parris Island, S.C. From 1947 to 1952 he was in Gulfport, Mississippi, as Director of Professional Education at the VA Hospital.

Dr. Blasko came to Washington as Chief of Psychiatric Training in the VA Central Office in 1952. In 1954 he left Washington to become Commissioner of the Connecticut State Department of Mental Health, where he served until 1957. He returned to Washington as chief of the VA psychiatric division in 1957. In 1959 he became Assistant Director of Psychiatry, Neurology and Psychology Service, moving up to Director in 1962. He held this post until he retired in June.

Dr. Blasko was awarded the Exceptional Service Award, the VA's highest honor, for his service with the VA and in recognition of his writing the VA's "Bill of Rights for Patients."

Dr. Blasko was a Fellow of the American Psychiatric Association, a member of the American Medical Association, the American Association of Military Surgeons, an Associate member of the Medical Society of the District of Columbia from 1964, and a member of the Washington Psychiatric Society. He was the author of many articles in the field of mental health.

He leaves his wife, Mrs. Frances Blasko, and a son, Dr. John C. Blasko, of Baltimore, Md.

Extradural and Subdural Empyemas—Jenkins et al

(Continued from page 478)

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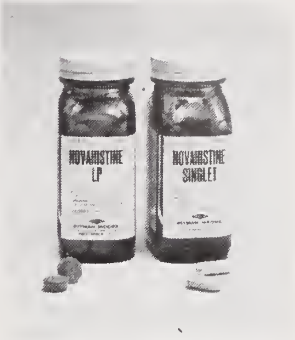

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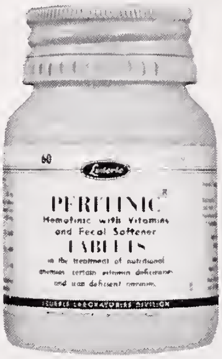

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* anticostive, *adj.* (*anti* opposed to + *costive* causing constipation.) Against constipation. Now isn't that a good idea in an iron-containing hematinic?

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(Available from the Superintendent of Documents, U. S. Government Printing Office, Washington, D. C. 20402. Single copies of most of these are available free upon request from the Public Health Service, Washington, D. C. 20201.)

Current Research in Chronic Airways Obstruction (PHS Publ. No. 1717). Contains 30 papers presented at the Ninth Aspen Emphysema Conference held in Aspen, Colorado, June 9–12, 1966. 400 pp. Price, $2.00.

Legal Aspects of PHS Medical Care—A Programmed Instruction Course (PHS Publ. No. 1468-A). Eli P. Bernzweig. Intended to show legal obligations and barriers that PHS physicians and other medical-health personnel face when they treat patients in facilities of the Public Health Service. Price, 55 cents. A reference booklet by the same title (PHS Publ. No. 1468) sells for 50 cents.

Leukemias, Lymphomas, and Multiple Myeloma (PHS Publ. No. 1768). Symptoms, treatment and research are discussed. Price, 10 cents.

Mental Retardation Film List. Prepared for the Division of Mental Retardation, Social and Rehabilitation Service, by the National Audiovisual Center, Atlanta, Ga. 30333, to provide a resource of selected audiovisuals for use in mental retardation educational programs. 60 pp.

The Prevention of Mental Retardation Through Control of Infectious Diseases. Edited by Heinz F. Eichenwald, M.D. (PHS Publ. No. 1692). Proceedings of a conference convened by the National Institute of Child Health and Human Development in 1966 to provide a forum of discussion and an opportunity for the exchange of information among 38 of the country's top experts in mental retardation and infectious diseases. 375 pp. Price, $2.50. Single copies available free from Public Information Branch, National Institute of Child Health and Human Development, Bethesda, Md. 20014.

Selected Films: Heart Disease, Cancer, and Stroke (PHS Publ. No. 1780). Compiled by The National Medical Audiovisual Center, Atlanta, Ga. 30333. 76 pp.

Selected References on Group Practice. An updated bibliography, the first of a series of materials to be developed on the subject. Prepared by the Group Practice Section, Health Economics Branch, Division of Medical Care Administration, Arlington, Va. 22203. 29 pp.

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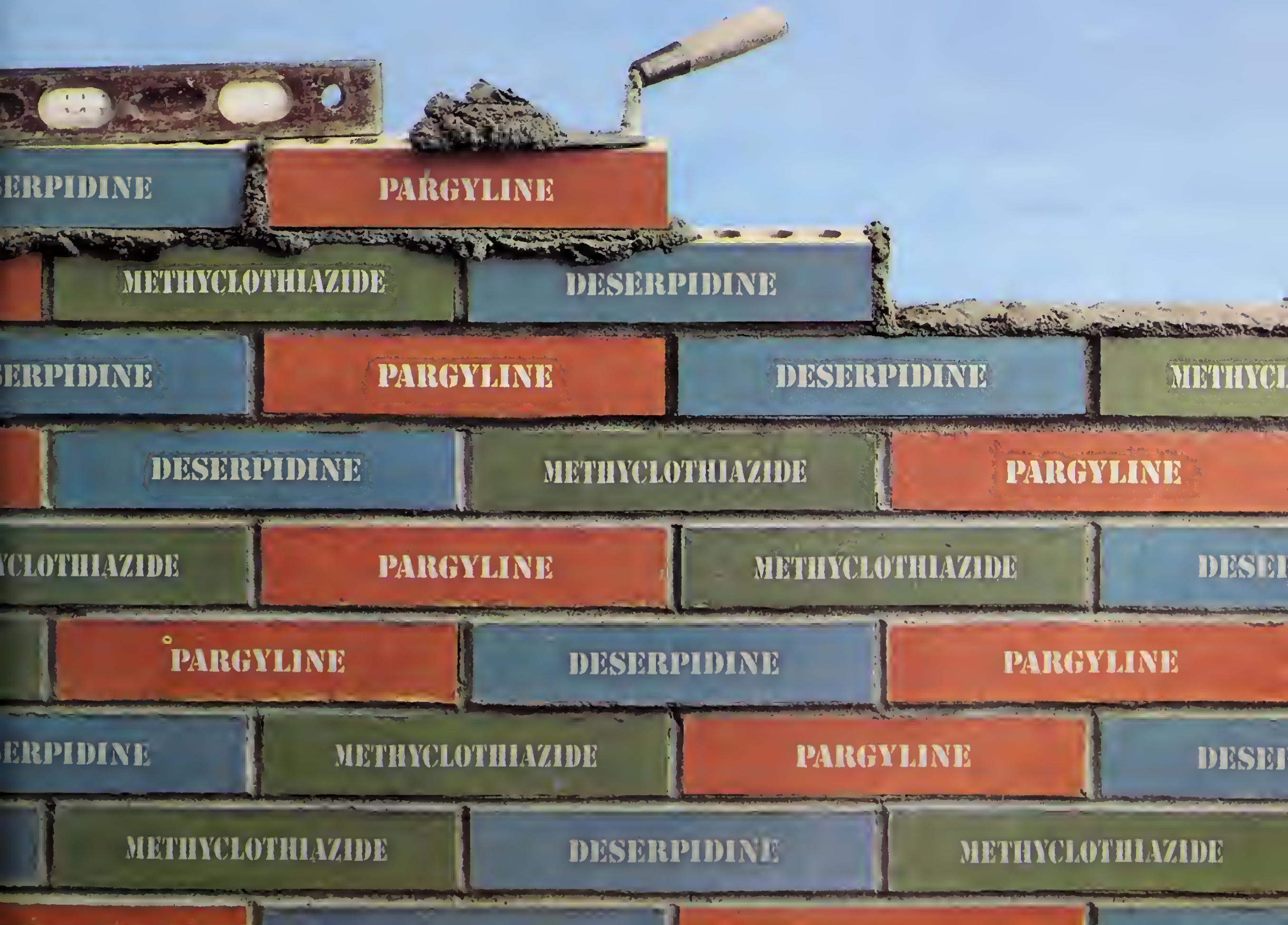
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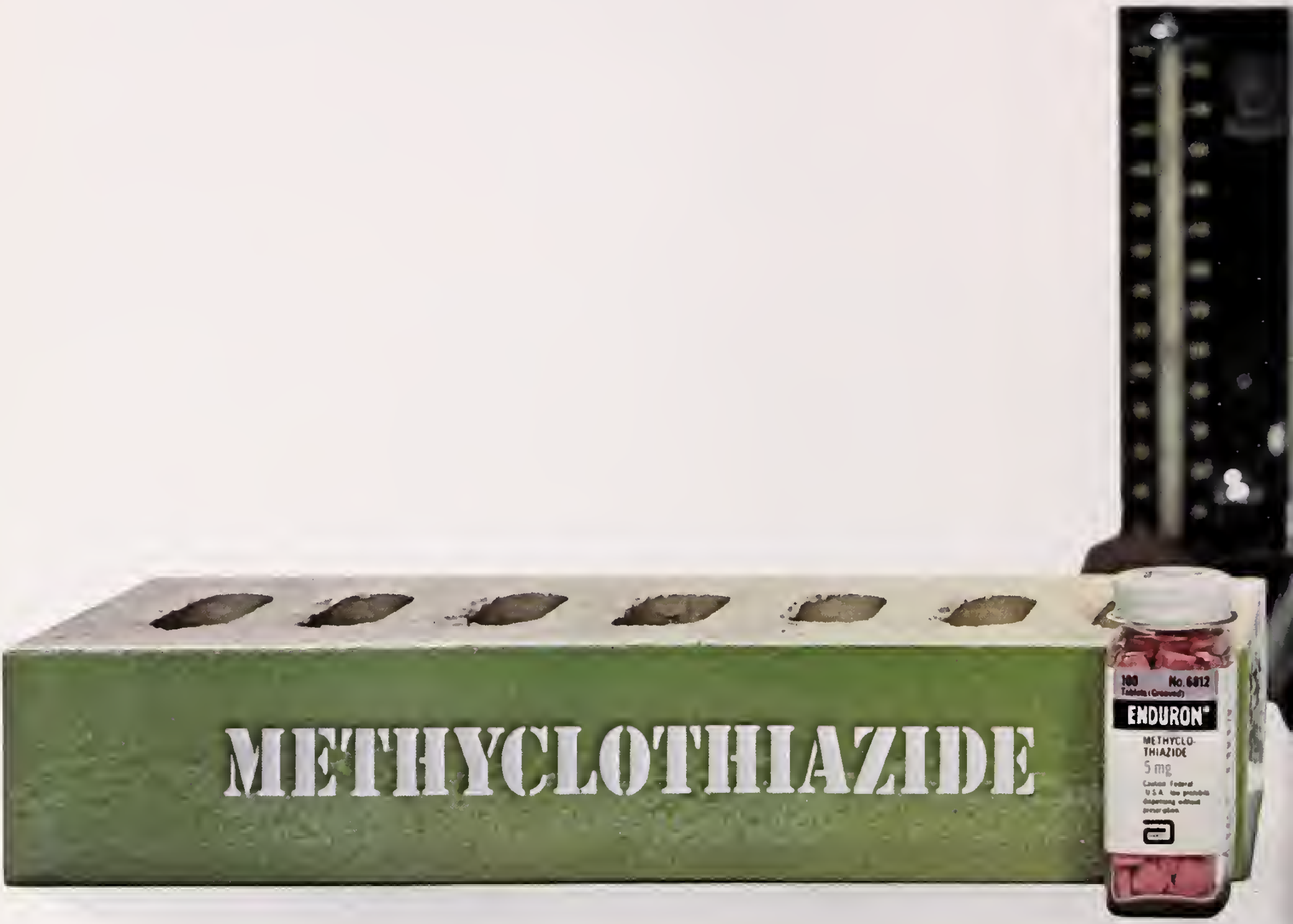
Enduron: (methyclothiazide) A basic building block for mild hypertensives



Excellent day-long Na$^+$ output, yet easy on the K$^+$

Enduron provides an excellent starting therapy. Your patient's sodium excretion is greatly enhanced. Yet potassium loss is low.

The therapeutic action is smooth, and persists for a full 24 hours. With Enduron you can prescribe convenient once-a-day dosage without skimping your patients on day-long thiazide effectiveness.

Of course, as with all thiazides, supplemental dietary potassium should also be considered.

Use Enduron as a basic therapy in patients with mild to moderate hypertension. A single 5-mg. tablet each day is ample in most cases.

Once a day, every day

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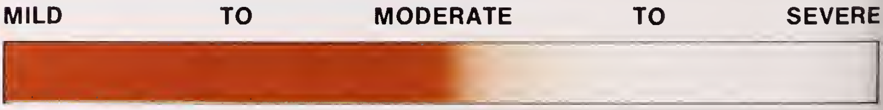

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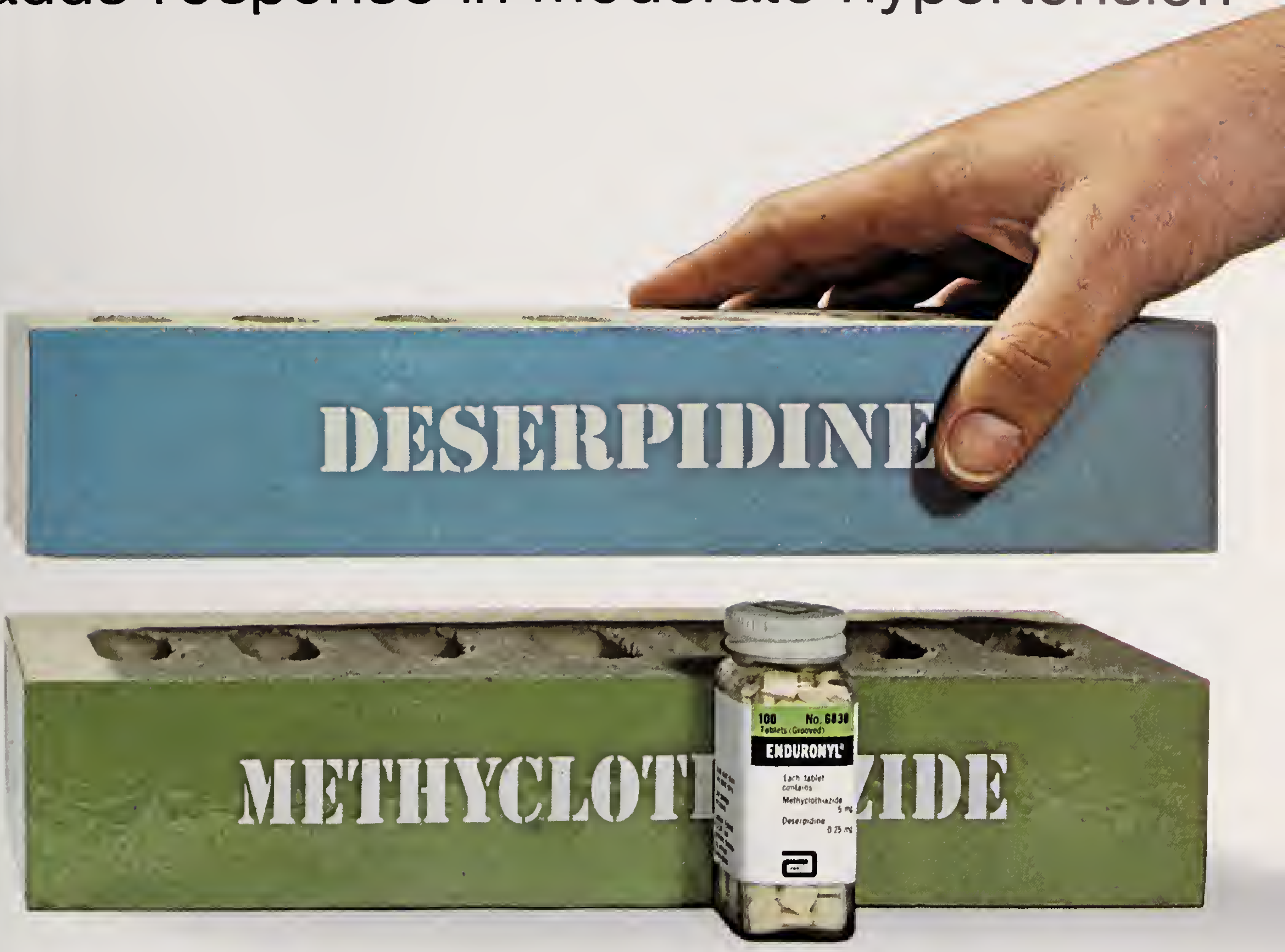
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ENDURONYL
Each tablet contains
Methyclothiazide 5 mg
Deserpidine 0.25 mg

Less frequent rauwolfia side effects than with reserpine

When you wish to build further response, consider shifting to Enduronyl.

Enduronyl adds a building block of *deserpidine*. This is a purified rauwolfia alkaloid available only from Abbott. It adds good antihypertensive and tranquilizing activity. Yet its incidence of untoward effects, particularly lethargy and depression, is lower than with reserpine.

Enduronyl is available plain or Forte. The latter provides its variation where most helpful, by doubling the deserpidine.

Use Enduronyl for patients throughout the broad range of mild to moderately severe hypertension.

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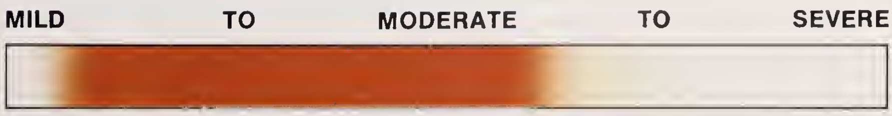
METHYCLOTHIAZIDE 5 mg. with
DESERPIDINE 0.25 mg. or (FORTE) 0.5 mg.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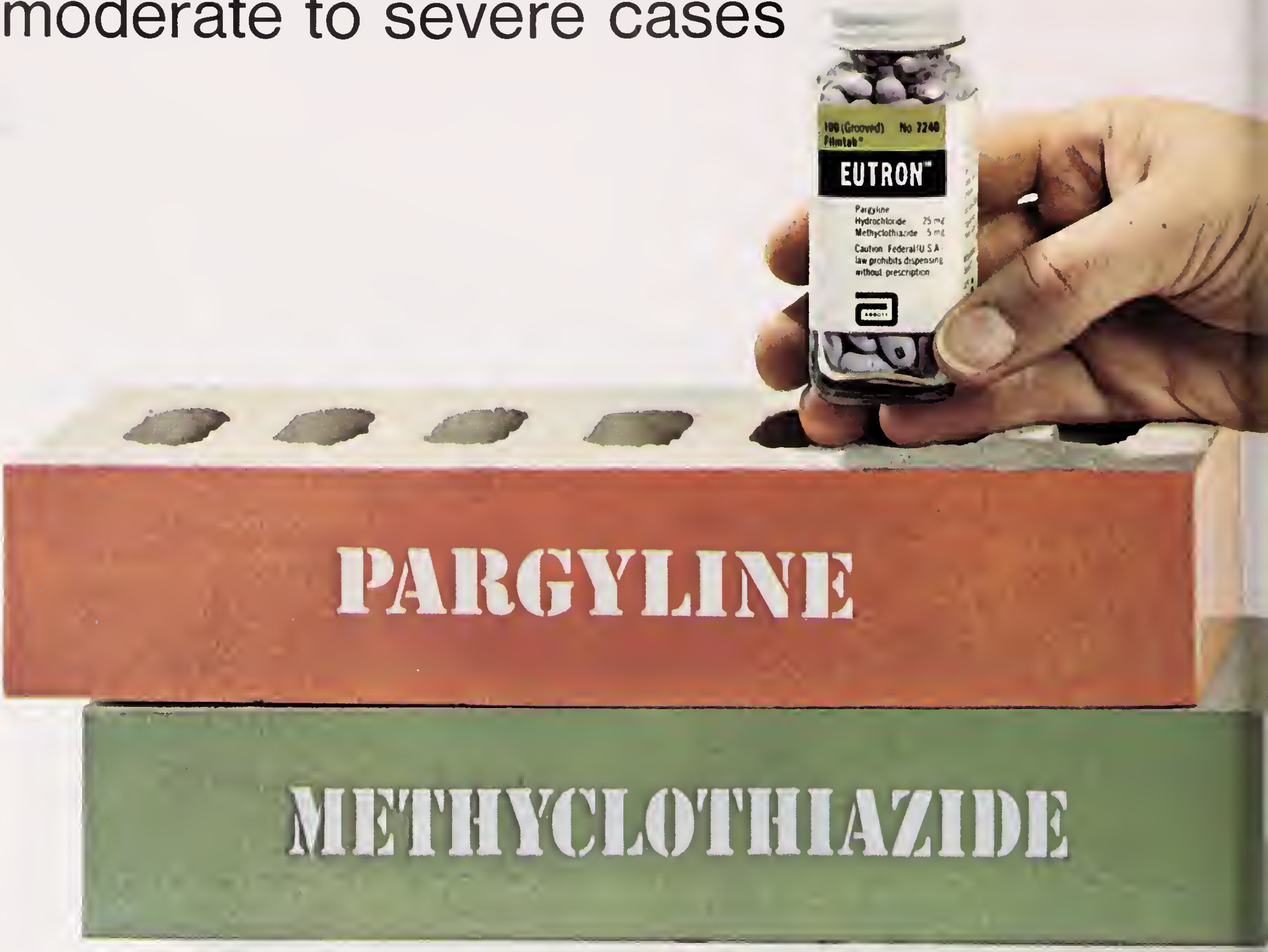
Eutron: A unique combination for handling moderate to severe cases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METHYCLOTHIAZIDE

Affords almost uniform diastolic reduction in all body positions

Eutron lowers diastolic pressures nearly equally, whether your patient is standing up or lying down.

Thus, in clinical trials, average *standing* diastolic readings were reduced from 112 pre-treatment to 90 post-treatment; *sitting* from 115 to 95; and *recumbent* from 112 to 94.

Note that following Eutron, the diastolic reductions were nearly alike in all three body positions.

Use Eutron for managing your moderate to severe cases. Its building blocks enhance each other; hence lesser doses often suffice.

Once a day, every day

EUTRON™

PARGYLINE HYDROCHLORIDE 25 mg.
with METHYCLOTHIAZIDE 5 mg.

MILD
TO
MODERATE
TO
SEVERE

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801094

ENDURON® | **ENDURONYL®**

METHYCLOTHIAZIDE

Each tablet contains
Methyclothiazide 5 mg. with
Deserpidine 0.25 mg. or 0.5 mg.

Indications: Edema and mild to moderate hypertension (Enduron), and mild to moderately severe hypertension (Enduronyl). More potent agents, if added, can be given at reduced dosage.

Contraindications: Sensitivity to thiazides; severe renal disease (except nephrosis) or shutdown; severe hepatic disease or impending hepatic coma (hepatic coma due to hypokalemia has been reported in patients on thiazides). Do not use Enduronyl in severe mental depression, suicidal tendencies, active peptic ulcer, or ulcerative colitis.

Warnings: Consider possible sensitivity where there is history of allergy or asthma. If added potassium is indicated, dietary supplementation is recommended. Reserve enteric-coated potassium tablets for cautious use only when necessary, as they may induce serious or fatal small bowel lesions (stenosis with or without ulceration), cause obstruction, hemorrhage, and perforation often requiring surgery; discontinue them immediately if abdominal pain, distention, nausea, vomiting, or g.i. bleeding occurs. Neither Enduron nor Enduronyl contains added potassium.

Precautions: Use thiazides cautiously in severe renal dysfunction, impaired hepatic function or progressive liver disease; also in pregnancy (bone marrow depression, thrombocytopenia, and altered carbohydrate metabolism have been reported in certain newborn). In surgery, thiazides may reduce response to vasopressors, and increase response to tubocurarine. Antihypertensive response may be enhanced following sympathectomy. Watch for electrolyte imbalance (e.g., hyponatremia) in all patients. In hypokalemia (especially in digitalized patients) give supplemental potassium. In hypochloremic alkalosis, give supplemental chloride.

Use rauwolfias with caution in patients with history of peptic ulcer. Rauwolfias with anesthetics may produce hypotension and bradycardia. Discontinue Enduronyl two weeks before elective surgery. Consider vagal blocking agents during emergency surgery. In epilepsy, adjust anticonvulsant dosage. In electroshock, shorten stimulus strength and duration. In occasional patients with depressive tendencies, rauwolfias may precipitate severe mental depression that usually disappears when drug is stopped.

Adverse Reactions: Thiazide reaction include blood dyscrasias (thrombocytopenia with purpura, agranulocytosis, aplastic anemia); elevation of BUN, serum uric acid or blood sugar; anorexia, nausea, vomiting, diarrhea, headache, dizziness, paresthesia, weakness, skin rash, photosensitivity, jaundice, symtomatic gout, and pancreatitis. Cutaneous vasculitis in the elderly has been reported with other thiazides. Adverse effects with deserpidine are qualitatively similar to those with reserpine, but their incidence is lower. These include nasal stuffiness, abdominal cramps or diarrhea, nausea, headache, weight gain, reduced libido and potency, peptic ulcer aggravation, epistaxis, skin eruption, asthma in susceptible patients, electrolyte imbalance, excessive salivation, and a reversible Parkinson's syndrome. Excessive drowsiness, fatigue, weakness, and nightmares may signal mental depression. Thrombocytopenia, purpura, and a symptom manifested by dull sensorium, deafness, uveitis, glaucoma, and optic atrophy are rare allergic reactions to other rauwolfias. Hypotension from antihypertensive agents may precipitate angina attacks in susceptible individuals. Usually adverse reactions disappear when drug is withdrawn.

EUTRON™ Each tablet contains
Pargyline Hydrochloride 25 mg.
with Methyclothiazide 5 mg.

Indications—Moderate to severe hypertension.

Contraindications—Pheochromocytoma, paranoid schizophrenia, hyperthyroidism and advanced renal failure. Not recommended in malignant hypertension, children under 12, pregnant patients.

Do not use with: centrally or peripherally acting sympathomimetic drugs; foods high in tyramine (e.g., aged and natural cheeses); parenteral reserpine or guanethidine; imipramine, amitriptyline, desipramine, nortriptyline or their analogues; other monoamine oxidase inhibitors; methyldopa or dopamine; separate Eutron and these agents by two weeks.

Sensitivity to thiazides; severe renal disease (except nephrosis) or shutdown; severe hepatic disease; impending hepatic coma from thiazide-induced hypokalemia.

Warnings—Patients: 1. No other drugs (particularly "cold preparations" and antihistamines), cheese or alcohol without physician's consent. 2. Promptly report orthostatic symptoms, severe headache, other unusual symptoms. 3. Angina pectoris or coronary artery disease patients must not increase physical activity with improved anginal symptoms or well-being.

Physicians: 1. Use antihistamines, hypnotics, sedatives, tranquilizers and narcotics (meperidine contraindicated) cautiously in reduced doses. 2. Stop Eutron two or more weeks before elective surgery; in emergency surgery reduce premedication (narcotics, sedatives, analgesics, etc.) to 1/4 to 1/5; carefully adjust anesthetic dosage to patient response. 3. Use cautiously in advanced renal failure. 4. Pargyline may induce hypoglycemia. 5. Consider possible sensitivity reactions when a history of allergy or asthma is present. 6. If potassium is indicated, dietary supplement is recommended; enteric-coated potassium tablets may induce serious or fatal small bowel lesions (stenosis with or without ulceration), cause obstruction, hemorrhage, and perforation frequently requiring surgery; discontinue medication immediately if abdominal pain, distention, nausea, vomiting or gastrointestinal bleeding occurs; Eutron does not contain added potassium. 7. Possible systemic lupus erythematosus has been reported for thiazides.

Precautions—Pargyline: Use cautiously at reduced dosage: caffeine, alcohol, antihistamines, barbiturates, chloral hydrate, other hypnotics, sedatives, tranquilizers, narcotics. Periodically do urinalyses, blood counts, liver function tests, etc. Use with caution in liver disease. Watch for orthostatic hypotension, especially in impaired circulation (e.g., angina pectoris, coronary artery disease, cerebral arteriosclerosis); also, augmented hypotension in concomitant febrile illnesses. Reduce or discontinue if hypotension is severe. In impaired renal function watch for cumulative drug effects, elevated BUN and other evidence of progressive renal failure; withdraw drug if these persist. In surgery increased central depressant response (hypotension and increased sedative effect) can be controlled by (1) discontinuing at least two weeks prior; (2) in emergency surgery lowering dose of premedication; (3) when necessary, administering a vasopressor. Do not use in hyperactive and hyperexcitable patients. Pargyline may unmask severe psychotic symptoms where emotional problems pre-exist. Use cautiously in Parkinsonism, especially with antiparkinsonian agents. In prolonged therapy, examine for change in color perception, visual fields, fundi and visual acuity. Also, prolonged therapy has made certain patients refractory to nerve blocking effects of local anesthetics.

Methyclothiazide: Use cautiously in severe renal dysfunction, impaired hepatic function or progressive liver disease; also in pregnancy (bone marrow depression, thrombocytopenia, and altered carbohydrate metabolism have been reported in certain newborn). In surgery thiazide may reduce vasopressor response and increase tubocurarine response. Antihypertensive response may be enhanced following sympathectomy. Watch for electrolyte imbalance (e.g., hyponatremia). Give supplemental chloride if hypochloremic alkalosis occurs and supplemental potassium if hypokalemia occurs (especially in digitalized patients). Thiazides may decrease serum P.B.I. without signs of thyroid disturbance.

Adverse Reactions — Pargyline: Orthostatic hypotension and associated symptoms, mild constipation, fluid retention, edema, dry mouth, sweating, increased appetite, arthralgia, nausea, vomiting, headache, insomnia, difficult in micturition, nightmares, impotence, delayed ejaculation, rash, purpura, weight gain, hyperexcitability, increased neuromuscular activity and other extrapyramidal symptoms. Drug fever is extremely rare. Reduction in blood sugar and hypoglycemic effects are possible. Congestive heart failure has been reported in a few patients with reduced cardiac reserve.

Methyclothiazide: Blood dyscrasias (thrombocytopenia with purpura, agranulocytosis, aplastic anemia); elevation of BUN, blood sugar or serum uric acid (gout may be induced); anorexia, nausea, vomiting, diarrhea, headache, dizziness, paresthesia, weakness, skin rash, photosensitivity, jaundice and pancreatitis. Cutaneous vasculitis in elderly patients has been reported with other thiazides.

If side effects are severe or persist, reduce dosage or withdraw drug. 804438R

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For profound calming effect in moderate to severe mental and emotional disturbances of everyday practice.

Before prescribing, see complete information, including adverse effects reported with phenothiazines and symptoms and treatment of overdosage, in SK&F literature or *PDR*. The following is a brief precautionary statement.

Contraindications: Comatose states or the presence of large amounts of C.N.S. depressants.

Precautions: Potentiation of C.N.S. depressants may occur (reduce dosage of such agents when used concomitantly). Use with caution in patients with chronic respiratory disorders. Antiemetic effect may mask overdosage of toxic drugs or obscure other conditions. Administer in pregnancy only when necessary. Because of possible drowsiness use cautiously and warn patients who operate vehicles or machinery.

Adverse Reactions: Drowsiness; dry mouth; nasal congestion; constipation; amenorrhea; miosis; mild fever; weight gain; hypotensive effects, sometimes severe with I.M. administration; epinephrine effects may be reversed; dermatological reactions; parkinsonism-like symptoms on high dosages (in rare instances, may persist); lactation and moderate breast engorgement (in females on high dosages); and less frequently, cholestatic jaundice (use cautiously in patients with liver disease). Adverse reactions occurring rarely, include: mydriasis; agranulocytosis; skin pigmentation; epithelial keratopathy; lenticular and corneal deposits (after prolonged substantial doses).

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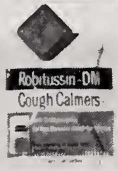
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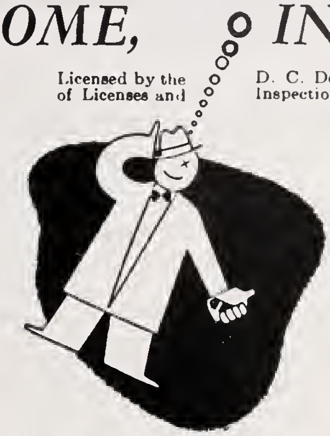

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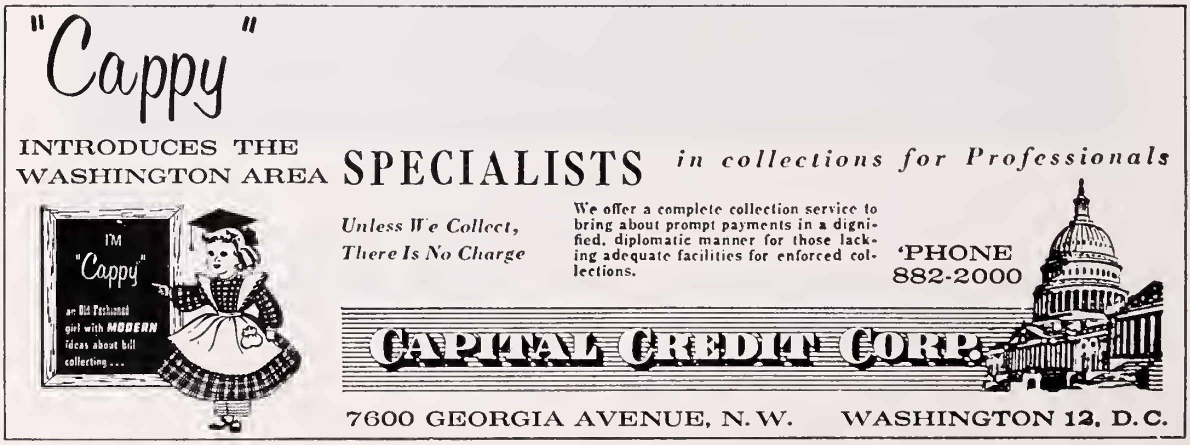

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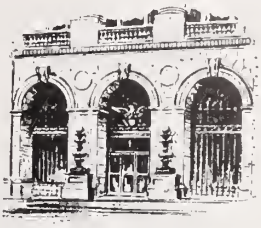

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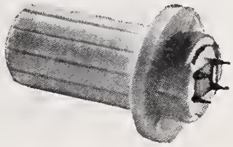

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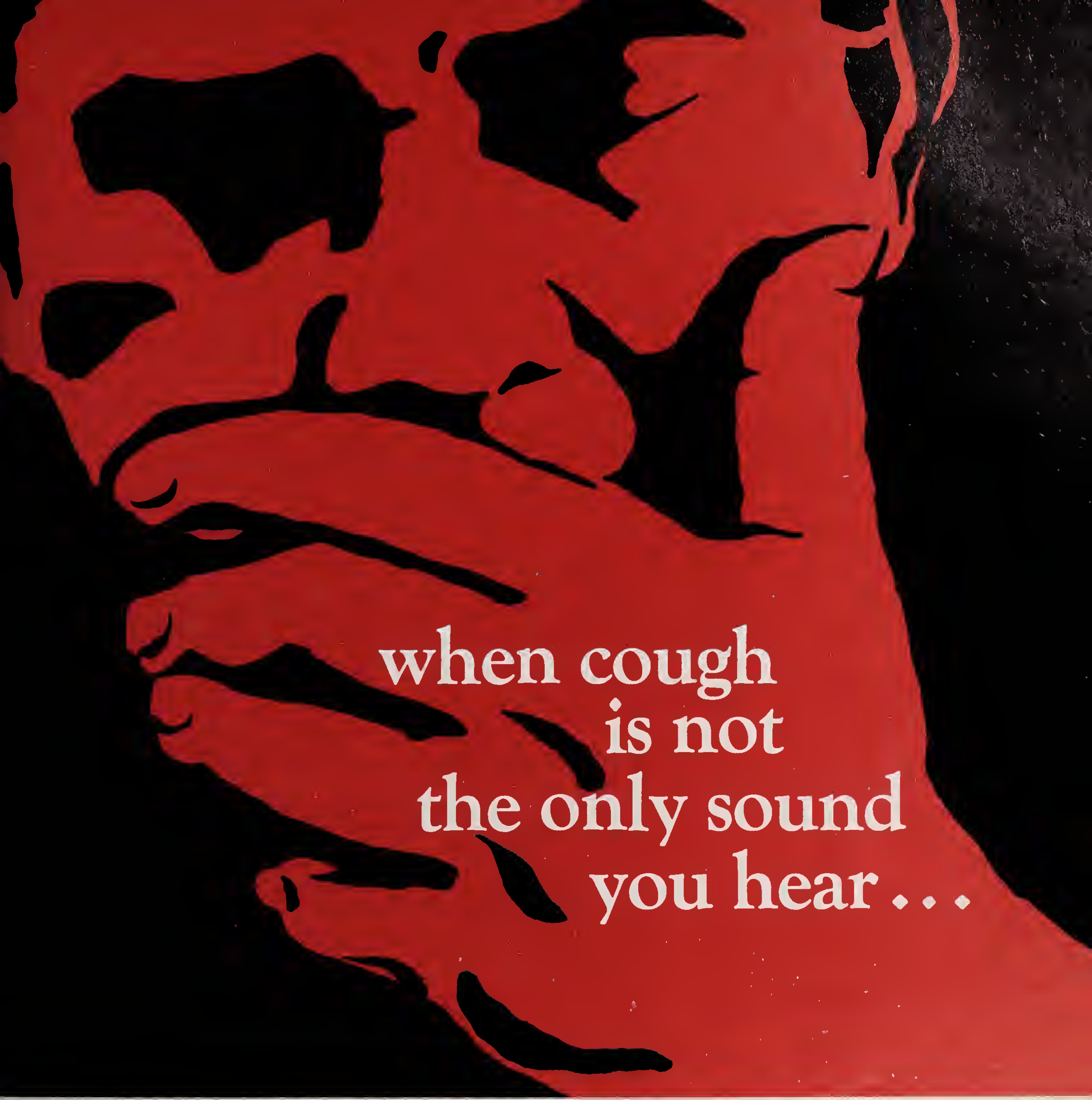

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was far superior to erythromycin, as was cloxacillin, a bactericidal agent, and of particular interest, "...bacteriostatic triacetyloleandomycin was as effective or perhaps superior to cloxacillin in preventing lethal (staphylococcal, phage type 80/81) infection."

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References: 1. Isenberg, H. D.: Clinical Evaluation of Laboratory Guidance to Antibiotic Therapy; Health Laboratory Science (July) 1967. 2. Saslaw, S., Carlisle, H. N.: Studies on Therapy of Staphylococcal Infections in Monkeys. 1. Comparison of Cloxacillin, Triacetyloleandomycin and Erythromycin. Proc. Soc. Exp. Biol. & Med.: Vol. 125, No. 4 (Aug.-Sept.) 1967.

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Warnings: Administer cautiously to patients who have previously exhibited a hypersensitivity reaction (e.g., blood dyscrasias, jaundice) to phenothiazines. Phenothiazines are capable of potentiating central nervous system depressants (e.g., anesthetics, opiates, alcohol, etc.) as well as atropine and phosphorus insecticides. During pregnancy, administer only when necessary.
Precautions: There have been infrequent reports of leukopenia and/or agranulocytosis and convulsive seizures. In epileptic patients, anticonvulsant medication should also be maintained. Pigmentary retinopathy may be avoided by remaining within the recommended limits of dosage. Administer cautiously to patients participating in activities requiring complete mental alertness (e.g., driving). Orthostatic hypotension is more common in females than in males. Do not use epinephrine in treating drug-induced hypotension. Daily doses in excess of 300 mg. should be used only in severe neuropsychiatric conditions.
Adverse Reactions: *Central Nervous System*—Drowsiness, especially with large doses, early in treatment; infrequently, pseudoparkinsonism and other extrapyramidal symptoms; nocturnal confusion, hyperactivity, lethargy, psychotic reactions, restlessness, and headache. *Autonomic Nervous System*—Dryness of mouth, blurred vision, constipation, nausea, vomiting, diarrhea, nasal stuffiness, and pallor. *Endocrine System*—Galactorrhea, breast engorgement, amenorrhea, inhibition of ejaculation, and peripheral edema. *Skin*—Dermatitis and skin eruptions of the urticarial type, photosensitivity. *Cardiovascular System*—Changes in the terminal portion of the electrocardiogram have been observed in some patients receiving the phenothiazine tranquilizers, including Mellaril (thioridazine hydrochloride). While there is no evidence at present that these changes are in any way precursors of any significant disturbance of cardiac rhythm, several sudden and unexpected deaths apparently due to cardiac arrest have occurred in patients previously showing electrocardiographic changes. The use of periodic electrocardiograms has been proposed but would appear to be of questionable value as a predictive device. *Other*—A single case described as parotid swelling.

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Precautions: In the elderly and debilitated, and in children over six, limit to smallest effective dosage (initially 10 mg or less per day) to preclude ataxia or oversedation, increasing gradually as needed and tolerated. Not recommended in children under six. Though generally not recommended, if combination therapy with other psychotropics seems indicated, carefully consider individual pharmacologic effects, particularly in use of potentiating drugs such as MAO inhibitors and phenothiazines. Observe usual precautions in presence of impaired renal or hepatic function. Paradoxical reactions (*e.g.*, excitement, stimulation and acute rage) have been reported in psychiatric patients and hyperactive aggressive children. Employ usual precautions in treatment of anxiety states with evidence of impending depression; suicidal tendencies may be present and protective measures necessary. Variable effects on blood coagulation have been reported very rarely in patients receiving the drug and oral anticoagulants; causal relationship has not been established clinically.

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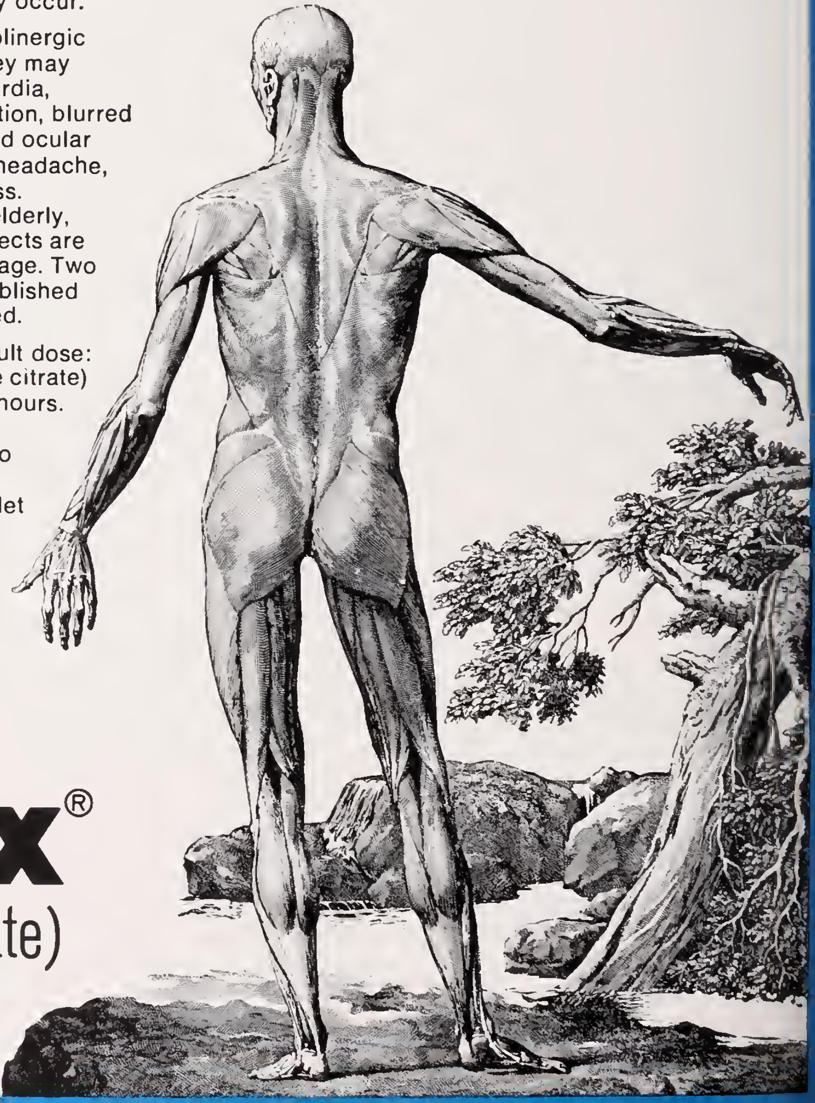

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References:

(1) Siver, R. H.: CMD, *21*:109, September 1954. (2) Frykman, H. H.: Minn. Med., *38*:19-27, January 1955. (3) McGivney, J.: Tex. State Jour. Med., *51*:16-18, January 1955. (4) Quehl, T. M.: Jour. of Florida Acad. Gen. Prac., *15*:15-16, October 1965. (5) Weekes, D. J.: N.Y. State Jour. Med., *58*:2672-2673, August 1958. (6) Weekes, D. J.: EENT Digest, *25*:47-59, December 1963. (7) Abbott, P. L.: Jour. Oral Surg., Anes., & Hosp. Dental Serv., 310-312, July 1961. (8) Rapoport, L. and Levine, W. I.: Oral Surg., Oral Med. & Oral Path., *20*:591-593, November 1965.

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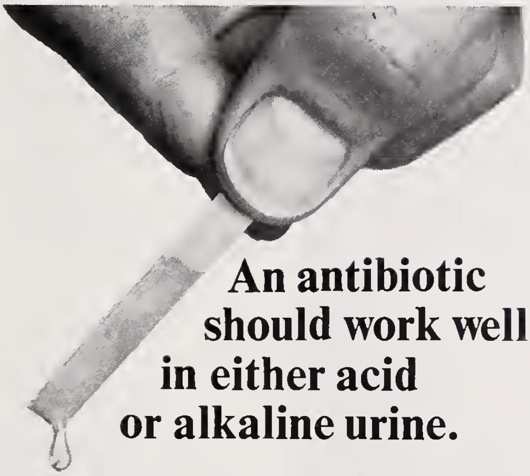

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Warning: In renal impairment, usual doses may lead to excessive accumulation and liver toxicity. Under such conditions, lower than usual doses are indicated, and, if therapy is prolonged, serum level determinations may be advisable. A photodynamic reaction to natural or artificial sunlight has been observed. Small amounts of drug and short exposure may produce an exaggerated sunburn reaction which may range from erythema to severe skin manifestations. In a smaller proportion, photoallergic reactions have been reported. Patients should avoid direct exposure to sunlight and discontinue drug at the first evidence of skin discomfort. Necessary subsequent courses of treatment with tetracyclines should be carefully observed.

Precautions: Overgrowth of nonsusceptible organisms may occur. Constant observation is essential. If new infections appear, appropriate measures should be taken. In infants, increased intracranial pressure with bulging fontanels has been observed. All signs and symptoms have disappeared rapidly upon cessation of treatment.

Side Effects: Gastrointestinal system – anorexia, nausea, vomiting, diarrhea, stomatitis, glossitis, enterocolitis, pruritus ani. Skin – maculopapular and erythematous rashes; a rare case of exfoliative dermatitis has been reported. Photosensitivity; onycholysis and discoloration of the nails (rare). Kidney – rise in BUN, apparently dose-related. Transient increase in urinary output, sometimes accompanied by thirst (rare). Hypersensitivity reactions—urticaria, angioneurotic edema, anaphylaxis. Teeth – dental staining (yellow-brown) in children of mothers given this drug during the latter half of pregnancy, and in children given the drug during the neonatal period, infancy and early childhood. Enamel hypoplasia has been seen in a few children. If adverse reaction or idiosyncrasy occurs, discontinue medication and institute appropriate therapy. Demethylchlortetracycline may form a stable calcium complex in any bone-forming tissue with no serious harmful effects reported thus far in humans.

Average Adult Daily Dosage: 150 mg q.i.d. or 300 mg b.i.d. Should be given 1 hour before or 2 hours after meals, since absorption is impaired by the concomitant administration of high calcium content drugs, foods and some dairy products. Treatment of streptococcal infections should continue for 10 days, even though symptoms have subsided.

Capsules: 150 mg; Tablets: film coated – 300 mg, 150 mg and 75 mg of demethylchlortetracycline HCl.

355-8

DECLOMYCIN®
DEMETHYLCHLORTETRACYCLINE

Lederle LEDERLE LABORATORIES, A Division of American Cyanamid Company, Pearl River, New York

This advertisement for TAO® (triacetyloleandomycin), published at the request of the Food and Drug Administration, replaces a recent one which the FDA regards as misleading.

The advertisement headlined "new evidence for TAO . . ." and emphasized that the drug is "for the frequently seen respiratory infection in the office and for a problem pathogen* in the hospital. *Staphylococcus aureus."

We emphasize that triacetyloleandomycin is to be used only for acute, severe bacterial infections where adequate sensitivity testing has demonstrated susceptibility to this drug and resistance to other less toxic agents. In view of the possible, but reversible, jaundice and hepatotoxicity of this drug, other less toxic agents should be used unless the organism is resistant to those agents, or in those cases where hypersensitivity precludes their use.

TAO is contraindicated in preexisting liver disease or dysfunction, and in individuals who have shown hypersensitivity to the drug.

The advertisement emphasized that no tooth staining has been reported after ten years of use of this antibiotic. The Food and Drug Administration regards this claim as an implied comparison suggesting that triacetyloleandomycin and tetracycline have a similar antibacterial spectrum of effectiveness, and that TAO has less toxic potential. Any such implication is not intended and, of course, would be invalid.

The advertisement referred to a research study in which patients were given triacetyloleandomycin prior to determining the susceptibility of the offending organism. Any suggestion that triacetyloleandomycin be used clinically without first determining susceptibility of the offending organism should be disregarded.

ROERIG Rx

J. B. ROERIG DIVISION
CHAS. PFIZER & CO., INC.
235 EAST 42nd STREET
NEW YORK, N.Y. 10017

TAO®(triacetyloleandomycin)
Brief Summary

INDICATIONS: Include streptococci, staphylococci, pneumococci and gonococci. Recommended for acute, severe infections where adequate sensitivity testing has demonstrated susceptibility to this antibiotic and resistance to less toxic agents.

CONTRAINDICATIONS: Contraindicated in pre-existing liver disease or dysfunction, and in individuals hypersensitive to the drug.

PRECAUTIONS: CAUTION: USE OF THIS DRUG MAY PRODUCE ALTERATIONS IN LIVER FUNCTION TESTS AND JAUNDICE. CLINICAL EXPERIENCE AVAILABLE THUS FAR INDICATES THAT THESE LIVER CHANGES WERE REVERSIBLE FOLLOWING DISCONTINUATION OF THE DRUG.

Not recommended for prophylaxis or in the treatment of infectious processes, which may require more than ten days continuous therapy. In view of the possible hepatotoxicity of this drug when therapy beyond ten days proves necessary, other less toxic agents should be used. If clinical judgment dictates continuation of therapy for longer periods, serial monitoring of liver profile is recommended, and the drug should be discontinued at the first evidence of any form of liver abnormality. When treating gonorrhea in which lesions of primary or secondary syphilis are suspected, proper diagnostic procedures, including dark-field examinations, should be followed. In other cases in which concomitant syphilis is suspected, monthly serological tests should be made for at least four months. When used in streptococcal infections, therapy should be continued for ten days to prevent the development of rheumatic fever or glomerulonephritis. The use of antibiotics may occasionally permit overgrowth of nonsusceptible organisms. A resistant infection or superinfection requires reevaluation of the patient's therapy. In the event such occurs with this drug the medication should be discontinued, and specific antibacterial and supportive therapy instituted.

ADVERSE REACTIONS: Although reactions of an allergic nature are infrequent and seldom severe, those of the anaphylactoid type have occurred on rare occasions.

ROERIG

J. B. ROERIG DIVISION
CHAS. PFIZER & CO., INC.
235 EAST 42nd STREET
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---

painful night
leg cramps...
respond to specific
therapy with
QUINAMM™
(Quinine sulfate, 260 mg.,
Aminophylline, 195 mg.)
THE BETTMAN ARCHIVE

Just one tablet at bedtime • Prevents painful night leg cramps • Permits restful sleep

How many of your patients stamp their feet at night and lose sleep because of painful leg cramps? Unless prompted, they usually fail to report this distressing condition and suffer needlessly.

One tablet of QUINAMM at bedtime usually controls distressing night cramps and permits restful sleep with the initial dose.

Prescribing information—Composition: Each white, beveled, compressed tablet contains: Quinine sulfate, 260 mg., Aminophylline, 195 mg. **Indications:** For the prevention and treatment of nocturnal and recumbency leg muscle cramps, including those associated with arthritis, diabetes, varicose veins, thrombophlebitis, arteriosclerosis and static foot deformities. **Contraindications:** QUINAMM is contraindicated in pregnancy because of its quinine content. **Side Effects/Precautions:** Aminophylline may produce intestinal cramps in some instances, and quinine may produce symptoms of cinchonism, such as tinnitus, dizziness, and gastrointestinal disturbance. Discontinue use if ringing in the ears, deafness, skin rash, or visual disturbances occur. **Dosage:** One tablet upon retiring. Where necessary, dosage may be increased to one tablet following the evening meal and one tablet upon retiring. **Supplied:** Bottles of 100 and 500 tablets.

THE NATIONAL DRUG COMPANY
DIVISION OF RICHARDSON MERRELL INC.
PHILADELPHIA, PENNSYLVANIA 19144

Before prescribing, see complete prescribing information in SK&F literature or *PDR*.

Contraindications: Glaucoma, prostatic hypertrophy, stenosing peptic ulcer, pyloroduodenal or bladder neck obstruction.

Precautions: Use cautiously in the presence of hypertension, hyperthyroidism, coronary artery disease; warn vehicle or machine operators of possible drowsiness.

Usage in Pregnancy: Use in pregnancy, nursing mothers and women who might bear children only when potential benefits have been weighed against possible hazards.

Note: The iodine in isopropamide iodide may alter PBI test results and will suppress I^{131} uptake; discontinue 'Ornade' one week before these tests.

Adverse Reactions: Drowsiness; excessive dryness of nose, throat or mouth; nervousness; insomnia. Other known possible adverse reactions of the individual ingredients: nausea, vomiting, diarrhea, rash, dizziness, fatigue, tightness of chest, abdominal pain, irritability, tachycardia, headache, incoordination, tremor, difficulty in urination. Thrombocytopenia, leukopenia and convulsions have been reported.

Supplied: Bottles of 50 capsules.

a stuffy nose
is no
laughing matter

Ornade® Trademark

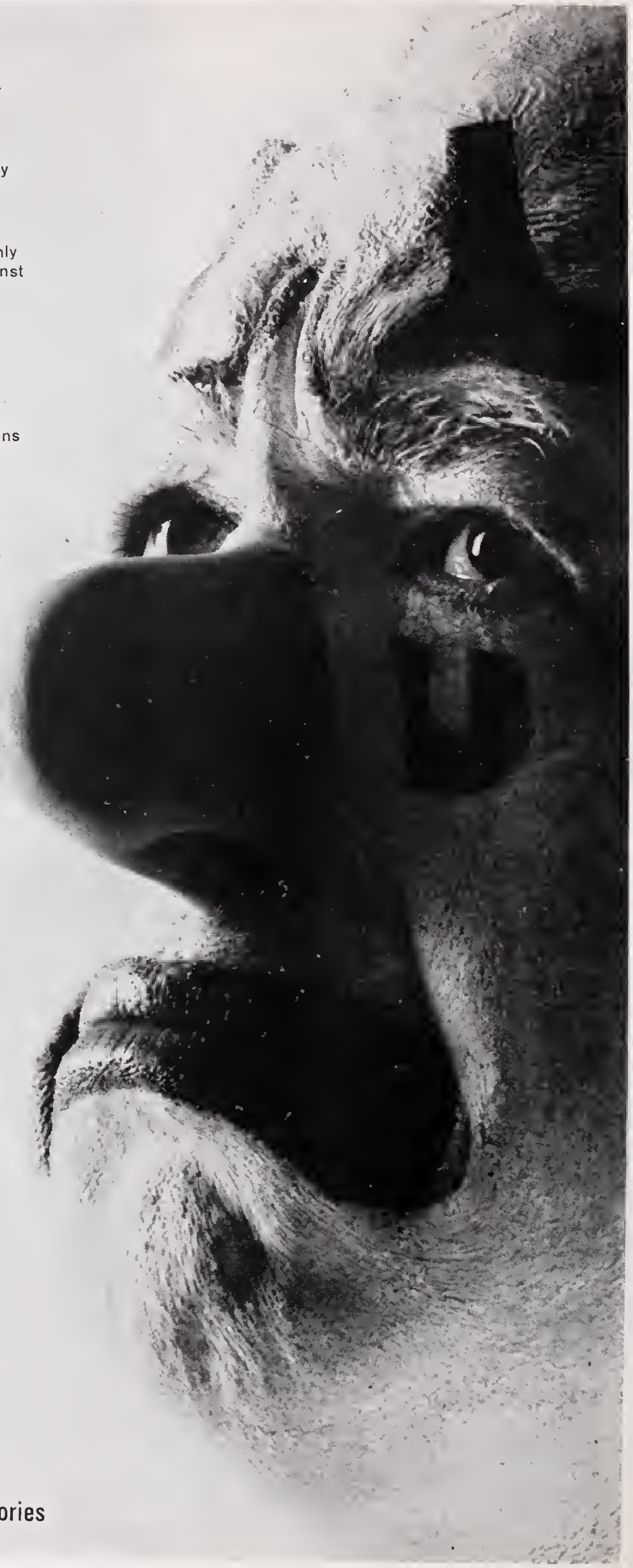
Each capsule contains 8 mg. of Teldrin® (brand of chlorpheniramine maleate), 50 mg. of phenylpropanolamine hydrochloride, and 2.5 mg. of isopropamide, as the iodide.

Spansule® Capsules

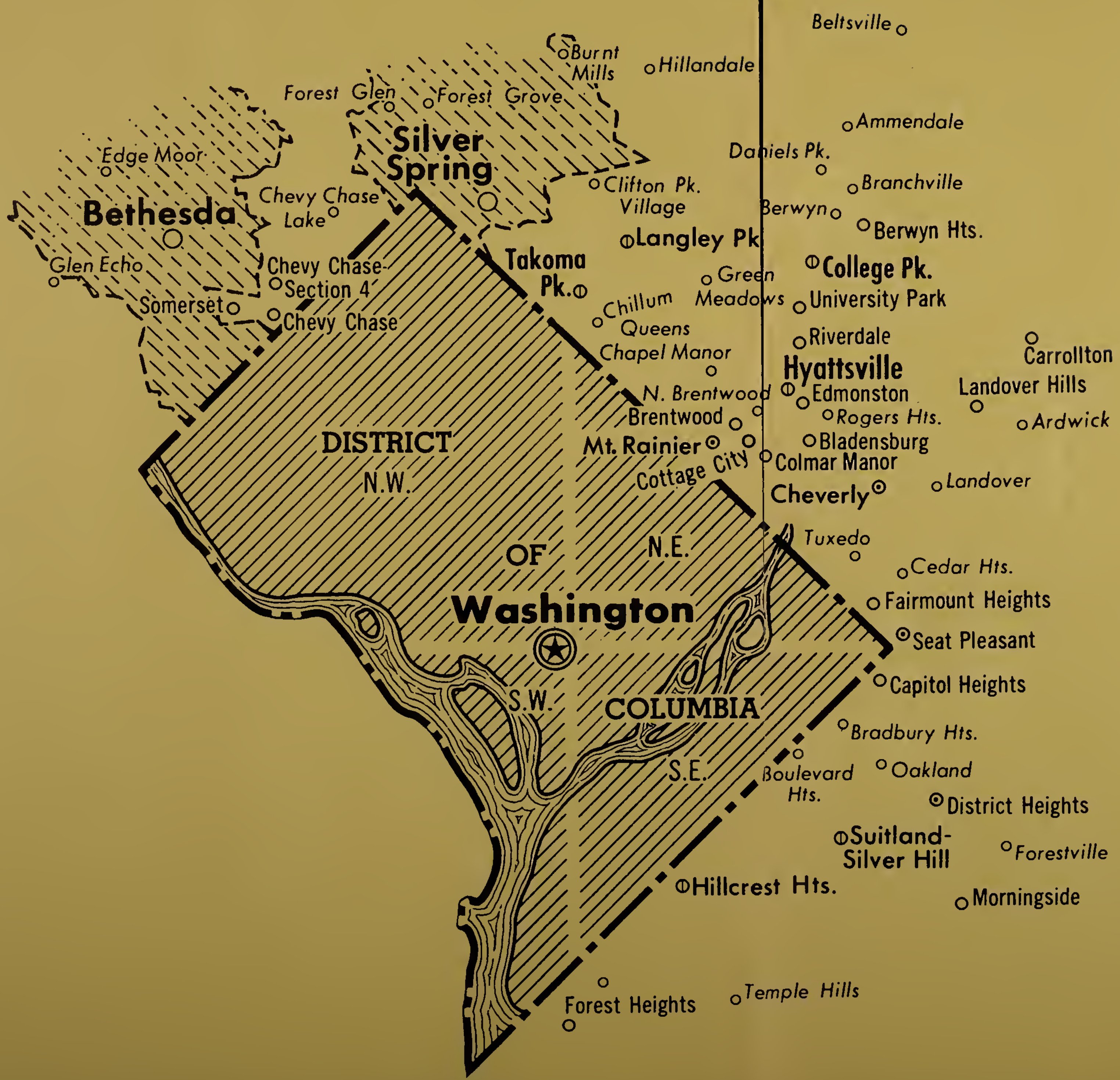
brand of sustained release capsules

each one can
give him all-day
or all-night relief
of nasal congestion

SK&F Smith Kline & French, Laboratories



ACHROMYCIN® WORKS HERE
TETRACYCLINE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Effectiveness: ACHROMYCIN Tetracycline is a crystalline broad-spectrum antibiotic which provides effective therapeutic activity against susceptible microorganisms.

Contraindication: History of hypersensitivity to tetracycline.

Warning: In renal impairment, usual doses may lead to excessive accumulation and liver toxicity. Under such conditions, lower than usual doses are indicated and, if therapy is prolonged, serum level determinations may be advisable. Some patients may develop a photodynamic reaction to natural or artificial sunlight. Those with a history of photosensitivity reactions should avoid direct exposure to sunlight while under treatment. Discontinue drug at first evidence of skin discomfort.

Precautions: Use may result in overgrowth of nonsusceptible organisms. Constant observation is essential. If new infections appear, take appropriate measures. Use of tetracycline during teeth development may cause discoloration of teeth.

Side Effects: Gastrointestinal system—anorexia, nausea, vomiting, diarrhea, stomatitis, glossitis, enterocolitis, pruritus ani. Skin—maculopapular and erythematous rashes (a case of exfoliative dermatitis has been reported); photosensitivity reaction, onycholysis and discoloration of nails (rare). Kidney—rise in BUN, apparently dose-related. Hypersensitivity reactions—urticaria, angioneurotic edema, anaphylaxis. In young infants, bulging fontanels have been reported following full therapeutic dosage. This symptom has disappeared rapidly when drug is discontinued. Teeth—dental staining (yellow-brown) in children of mothers given tetracycline during the latter half of pregnancy, and in children given the drug during the neonatal period, infancy and early childhood. Enamel hypoplasia has been seen in a few children. Blood—anemia, thrombocytopenic purpura, neutropenia, eosinophilia. Liver—cholestasis (rare), usually at high dosage. Tetracycline may form a stable calcium complex in bone-forming tissue. If adverse reaction or idiosyncrasy occurs, discontinue medication and institute appropriate therapy.

Average Adult Daily Dosage: One Gm. per day, in 4 divided doses of 250 mg. each. Should be given 1 hour before or 2 hours after meals, since absorption is impaired by the concomitant administration of high calcium content drugs, foods and some dairy products. Treatment of streptococcal infections should continue for 10 days, even though symptoms have subsided.

ACHROMYCIN® WORKS HERE
TETRACYCLINE

Actually ACHROMYCIN works wherever the infection is due to an organism your lab cultures have shown to be tetracycline-sensitive. No other tetracycline—or analogue—can assure you of quicker or more effective antibiotic action. After all, isn't that what you want for your patient?

ACHROMYCIN was a pioneer in tetracycline therapy. Seventeen years of experience have shown that when the respiratory system, the genitourinary system, or the skin and soft tissue require treatment for infection probably caused by any of a number of sensitive strains, ACHROMYCIN is a wise choice for therapy while awaiting test results.

Available in 31 useful forms to meet your situational needs.

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Book Reviews

Infectious Diseases of Children, 4th ed. Saul Krugman, M.D., and Robert Ward, M.D. Price, $16.50. Pp. 428, with 69 figures and 9 color plates (including a gatefold insert). St. Louis: Mosby, 1968.

Greeting a new edition of "Krugman and Ward" is like welcoming an old friend to your living room. You note immediately that he is still sprightly and well groomed. A few inquiries let you know he has kept up to date. (References and text have been revised to include the results of research through 1967.) Even some of his minor faults of the past have been corrected. (The incidence of clinical herpes simplex virus infections is recognized as being as high as 12 per cent, rather than less the 1 per cent previously reported.)

But it is the old virtues, rather than the changes, which make the new edition, like its predecessors, one of the few truly great books in clinical medicine. The authors draw on a broad personal involvement with research and clinical practice, combine it with a critical evaluation of other research, and digest and present the material in a format that is immensely readable and pedagogically sound, whether the reader is a medical student or a seasoned clinician.

Each of the first 28 chapters is devoted to a disease entity, sometimes organized by etiologic agent—enteroviral infections, sometimes by clinical syndrome—acute respiratory disease. The descriptions of the clinical course of the illnesses are masterpieces of lucidity and conciseness. The clues to differential diagnosis, especially the final chapter on Diagnosis of Acute Exanthematous Diseases, are alone worth the price of the book. To these are added the pertinent up-to-date knowledge of etiology, pathogenesis, pathology, and prevention.

The authors carefully distinguish fact from opinion but do not hesitate to state well reasoned opinion when facts alone are insufficient to support important clinical decisions.

Any physician caring for children will be better equipped after reading or rereading this book and, what is more, he will probably enjoy the experience thoroughly.

A. Frederick North, Jr., M.D.

Pediatric Therapy, 3rd ed. Harry C. Shirkey, B.S. (Pharm.), M.D., F.A.A.P., with 89 contributors. Price, $25.00. Pp. 1294, with 351 illustrations. St. Louis: Mosby, 1968.

Every physician involved in the medical care of children should have a current reference of therapy. Shirkey's "Pediatric Therapy," 1968–1969, has been reëdited for its third time in as many years. With all of its chapters being updated, it represents the most current compendium of pediatric therapy available to the practicing physician. On the whole it is as good as any textbook on therapy for children. It has the advantage that it is the most up-to-date.

The outstanding features of the book are:

1. Excellent sections on poisonings and drug dosages which are easily identified in the text. The poisons are listed in table form with approximate minimal lethal dosages, principal effects and symptoms, and specific treatment clearly outlined. This section appears as a pink insert in the text. The table of drugs is similar to a table by the author which appears in the "Textbook of Pediatrics" by W. E. Nelson, edition 8. It has the advantage of being revised and enlarged and is easily identified within the text by virtue of being printed on blue paper.

2. The section on the treatment of diseases of various organ systems is excellent. The various chapters are written by outstanding subspecialists in their respective fields.

3. There is a very good chapter on the chronically ill child which outlines the role of the physician as leader of a "team." As leader of the "team" he must integrate the skills and services of the nurse, the social worker, occupational therapist, recreational therapist, teacher, speech and hearing therapist, psychologist, dietitian, etc. to provide comprehensive care for the child. This chapter should be valuable to young physicians preparing themselves for a career in pediatrics.

As with any book that attempts to deal with the skin and its contents, there are certain weaknesses. The section on parenteral fluid therapy is weak and needs revision. In a textbook devoted to pediatric therapy a much stronger section on parenteral fluid therapy is warranted.

All in all, this is an excellent reference book of pediatric therapy.

Lewis P. Scott, M.D.

When it's more than a bad cold

your patient can feel better while he's getting better

Achrocidin®

Tetracycline HCl—Antihistamine—Analgesic Compound

Each tablet contains: ACHROMYCIN® Tetracycline HCl 125 mg.; Phenacetin 120 mg.; Caffeine 30 mg.; Salicylamide 150 mg.; Chlorothen citrate 25 mg.

In tetracycline-sensitive bacterial infection complicating respiratory allergy, ACHROCIDIN brings the treatment together in a single prescription—prompt relief of headache and congestion together with effective control of the organisms frequently responsible for complications leading to prolonged disability in the susceptible patient.

For children and elderly patients you may prefer caffeine-free ACHROCIDIN Syrup. Each 5 cc contains: ACHROMYCIN (Tetracycline) equivalent to Tetracycline HCl 125 mg.; Phenacetin 120 mg.; Salicylamide 150 mg.; Ascorbic Acid (C) 25 mg.; Pyrilamine Maleate 15 mg.

Contraindications: Hypersensitivity to any component.

Warning: In renal impairment, since liver toxicity is possible, lower doses are indicated; during prolonged therapy consider serum level determinations. Photodynamic reaction to sunlight may occur in hypersensitive persons. Photosensitive individuals should avoid exposure; discontinue treatment if skin discomfort occurs.

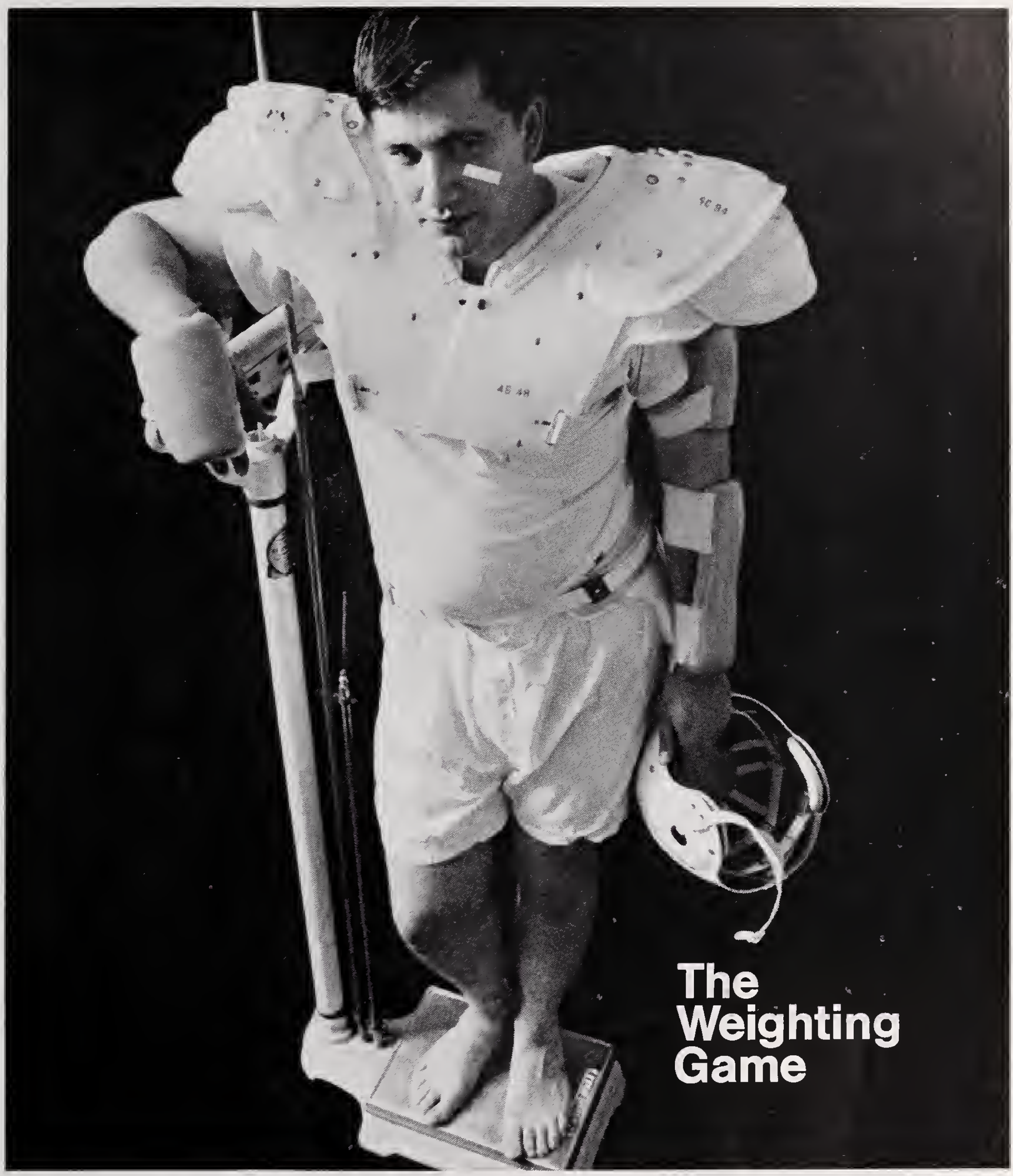
Precautions: Drowsiness, anorexia, slight gastric distress can occur. In excessive drowsiness, consider longer dosage intervals. Persons on full dosage should not operate vehicles. Nonsusceptible organisms may overgrow; treat superinfection appropriately. Treat beta-hemolytic streptococcal infections at least 10 days to help prevent rheumatic fever or acute glomerulonephritis. Tetracycline may form a stable calcium complex in bone-forming tissue and may cause dental staining during tooth development (last half of pregnancy, neonatal period, infancy, early childhood).

Adverse Reactions: *Gastrointestinal*—anorexia, nausea, vomiting, diarrhea, stomatitis, glossitis, enterocolitis, pruritus ani. *Skin*—maculopapular and erythematous rashes; exfoliative dermatitis; photosensitivity; onycholysis, nail discoloration. *Kidney*—dose-related rise in BUN. *Hypersensitivity* reactions—urticaria, angioneurotic edema, anaphylaxis. *Intracranial*—bulging fontanels in young infants. *Teeth*—yellow-brown staining; enamel hypoplasia. *Blood*—anemia, thrombocytopenic purpura, neutropenia, eosinophilia. *Liver*—cholestasis at high dosage.

Upon adverse reaction, stop medication and treat appropriately.

Lederle

348-8



Too often athletes take off too much too soon. They're using fad stuff and crash diets.

They don't seem to realize that a speed-up in reducing can cause a slow-down in producing.

In the long run, there's really no fast way.

Losing weight is a growing problem. Here's where you, the professional, can help. Educate. Inform. Explain to parents and team members the risks involved with quickie solutions. Tell them about a planned program of diet, exercise, proper rest and will power.

Project Weight Watch can help, too. We have a complete portfolio of materials and suggested diets available, free. The diets are a realistic balance of the 4 food groups—meat, breads and cereals, fruits and vegetables and dairy foods. The kind of diets you'd probably suggest yourself. It'll only take a minute to send for them.

PROJECT WEIGHT WATCH
FACTS, NOT FADS

Plan a losing strategy today.

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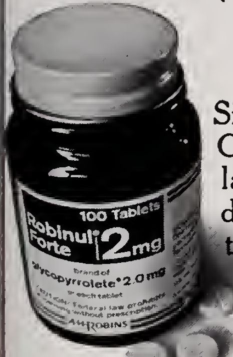
Summation:
In addition to its primary indications for duodenal and gastric ulcer, Robinul Forte (glycopyrrolate) is indicated for other G-I conditions that may benefit from anticholinergic therapy. Robinul-PH Forte (glycopyrrolate 2 mg. with phenobarbital) is indicated when these situations are complicated by mild anxiety and tension.
Contraindications: Glaucoma, urinary bladder neck obstruction, pyloric obstruction, stenosis with significant gastric retention, prostatic hypertrophy, duodenal obstruction, cardiospasm (megaesophagus), and achalasia of the esophagus, and in the case of Robinul-PH Forte, sensitivity to phenobarbital.
Precautions: Administer with caution in the presence of incipient glaucoma.
Adverse Reactions: Dryness of the mouth, blurred vision, urinary difficulties, and constipation are rarely troublesome and may generally be controlled by reduction of dosage. Other side effects associated with the use of anticholinergic drugs include tachycardia, palpitation, dilatation of the pupil, increased ocular tension, weakness, nausea, vomiting, headache, dizziness, drowsiness, and rash.
Dosage: Should be adjusted according to individual patient response. Average and maximum recommended dose is 1 tablet three times a day; in the a.m., early p.m., and at bedtime. See product literature for full prescribing information. Supply: Robinul (glycopyrrolate 1 mg.); Robinul Forte (glycopyrrolate 2 mg.); Robinul-PH (glycopyrrolate 1 mg.) with phenobarbital 16.2 mg. (Warning: may be habit forming); Robinul-PH Forte (glycopyrrolate 2 mg.) with phenobarbital 16.2 mg. (Warning: may be habit forming), in bottles of 100 and 500 tablets. A. H. Robins Company, Richmond, Va. 23220.

A·H·ROBINS

You be the judge, Docto

In peptic ulcer therapy, won't you give Robinul® Forte a Fair Trial?

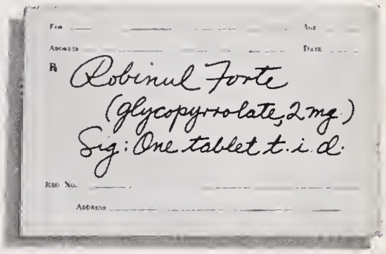
(glycopyrrolate)



Six years ago the A. H. Robins Company introduced glycopyrrolate, a unique anticholinergic agent described in the prescribing literature as more closely approaching the ideal compound for controlling gastric hyperacidity and hypermotility of the G-I tract. Although glycopyrrolate (Robinul Forte) found good acceptance among numerous physicians, many others just didn't seem to want to give it a try, probably because the anticholinergic they were already using was giving acceptable results.

However, we believe you'll agree there's always room for a better anticholinergic. This is why we're asking you to give Robinul Forte a fair trial. Robinul Forte exerts a highly specific antisecretory action and marked inhibitory effect on intestinal tone. We're convinced you'll agree that this is indeed an outstanding drug when you observe its outstanding suppression of ulcer symptoms. Furthermore, it is unique in that it reduces intestinal tone, yet has little or no effect on peristalsis. In addition, the incidence of the more bothersome peripheral side effects is low.

No longer does the physician have to look for extreme dry mouth as the measure of his anticholinergic's effectiveness. The only way we can demonstrate to you firsthand the efficacy of glycopyrrolate (Robinul Forte) is for you to try it in your practice. That's why we're asking you to give it a Fair Trial. How can you give it a Fair Trial? You do it this way:



℞ Robinul Forte
(glycopyrrolate, 2 mg.)
Sig: One tablet t.i.d.

First: When you see your very next ulcer patient, write him a script as shown.

Next: Wait 10 days or until your patient comes in for his next appointment and get his "verdict" as to how he feels. Examine all the evidence and make your evaluation of his condition.

Finally: Render **your** verdict. If it's "significant improvement and marked relief of symptoms," then we believe you'll agree that Robinul Forte has proved its worth, and we rest our case. If not, consider our case for Robinul Forte closed. That's a Fair Trial, Doctor, and it's all we ask.

Robinul® Forte

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For brief summary see next page.

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Contraindications: Edema; danger of cardiac decompensation; history or symptoms of peptic ulcer; renal, hepatic or cardiac damage; history of drug allergy; history of blood dyscrasia. The drug should not be given when the patient is senile or when other potent drugs are given concurrently.

Warning: This drug is an analog of phenylbutazone; sensitive patients may be cross-reactive. If coumarin-type anticoagulants are given simultaneously, watch for excessive increase in prothrombin time. Instances of severe bleeding have occurred. Persistent or severe dyspepsia may indicate peptic ulcer; perform upper gastrointestinal x-ray diagnostic tests if drug is continued. Pyrazole compounds may potentiate the pharmacologic action of sulfonylurea, sulfonamide-type agents and insulin. Carefully observe patients receiving such therapy. Use with caution in the first trimester of pregnancy, and in patients with thyroid disease.

Precautions: Before prescribing, carefully select patients, avoiding those responsive to routine measures as well as contraindicated patients. Obtain a detailed history and a complete physical and laboratory examination, including a blood count. The patients should not exceed recommended dosage, should be closely supervised and should be warned to discontinue the drug and report immediately if fever, sore throat, or mouth lesions (symptoms of blood dyscrasia), sudden weight gain (water retention), skin reactions, black or tarry stools or other evidence of intestinal hemorrhage occur. Make complete blood counts at weekly intervals during early therapy and at 2-week intervals thereafter. Discontinue the drug immediately and institute countermeasures if the white count changes significantly, granulocytes decrease, or immature forms appear. Use greater care in the elderly and in hypertensives.

Adverse Reactions: The more common are nausea and edema. Swelling of the ankles or face may be minimized by withholding dietary salt, reduction in dosage or use of diuretics. In elderly patients and in those with hypertension, the drug should be discontinued with the appearance of edema. The drug has been associated with peptic ulcer and may reactivate a latent peptic ulcer. The patient should be instructed to take doses immediately after meals or with milk to minimize gastric upset. Drug rash occasionally occurs. If it does, promptly discontinue the drug. Agranulocytosis, exfoliative dermatitis, Stevens-Johnson syndrome, Lyell's syndrome (toxic necrotizing epidermolysis), or a generalized allergic reaction similar to a serum sickness syndrome may occur and require permanent withdrawal of medication. Agranulocytosis can occur suddenly in spite of regular, repeated normal white counts. Stomatitis, salivary gland enlargement, vomiting, vertigo and languor may occur. Leukemia and leukemoid reactions have been reported. While not definitely attributable to the drug, a causal relationship cannot be excluded. Thrombocytopenic purpura and aplastic anemia may occur. Confusional states, agitation, headache, blurred vision, optic neuritis and transient hearing loss have been reported, as have hyperglycemia, hepatitis, jaundice, hypersensitivity angiitis, pericarditis and several cases of anuria and hematuria. With long-term use, reversible thyroid hyperplasia may occur infrequently. Moderate lowering of the red cell count due to hemodilution may occur.

Dosage in Osteoarthritis: Initial: 3 to 6 tablets daily in divided doses. Usually unnecessary to exceed 4 tablets daily. A trial period of one week is considered adequate to determine the therapeutic effect of the drug. Maintenance: Effective level often achieved with 1 or 2 tablets daily, should not exceed 4 tablets daily.

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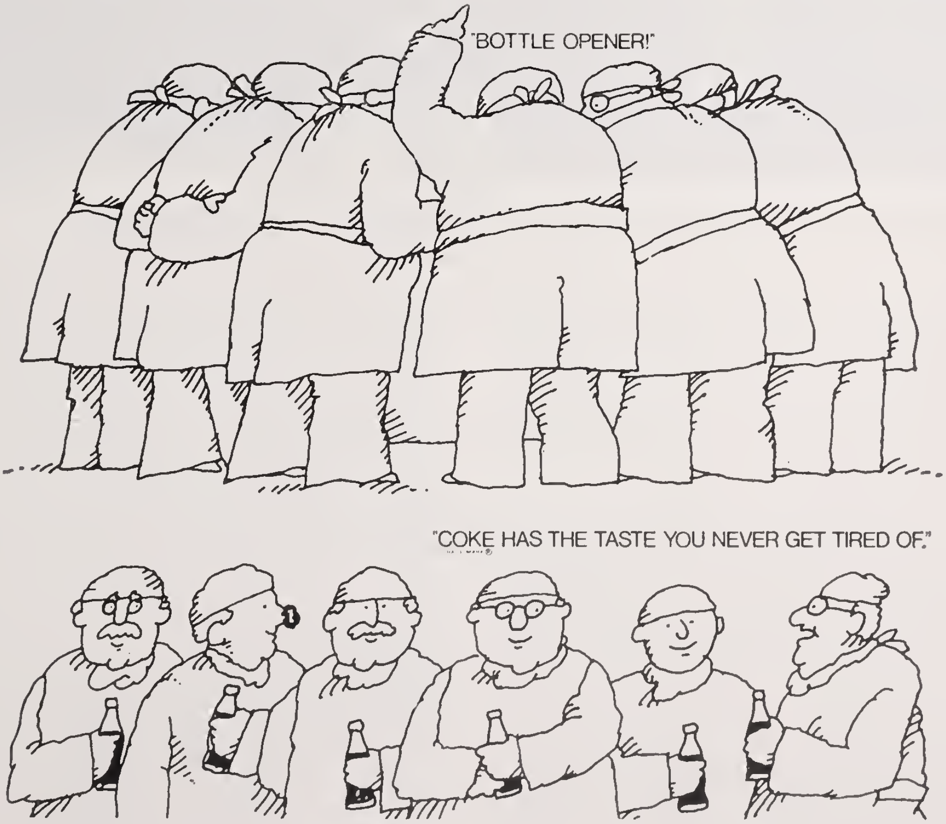
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Contraindication: Penicillin hypersensitivity.

Warnings: In rare instances, penicillin may cause acute anaphylaxis which may prove fatal unless promptly controlled. This type of reaction appears more frequently in patients with a history of sensitivity reactions to penicillin or with bronchial asthma or other allergies. Resuscitative drugs should be readily available. These include epinephrine and pressor drugs (as well as oxygen for inhalation) for immediate allergic manifestations and antihistamines and corticosteroids for delayed effects.

Precautions: Use cautiously, if at all, in a patient with a strongly positive history of allergy.

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In suspected staphylococcus infections, proper laboratory studies (including sensitivity tests) should be performed.

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Penicillin is a substance of low toxicity, but it possesses a significant index of sensitization. The following hypersensitivity reactions have been reported: skin rashes ranging from maculopapular eruptions to exfoliative dermatitis; urticaria; and reactions resembling serum sickness, including chills, fever, edema, arthralgia, and prostration. Severe and often fatal anaphylaxis has occurred (see Warnings). Hemolytic anemia, leukopenia, thrombocytopenia, and nephropathy are rarely observed side-effects and are usually associated with high parenteral dosage.

Administration and Dosage: Usual dosage range, 125 mg. (200,000 units) three times a day to 500 mg. (800,000 units) every four hours. For infants, 50 mg. per Kg. per day divided into three doses.

See package literature for detailed dosage instructions for prophylaxis of streptococcus infections, surgery, gonorrhea, and severe infections.

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MEDICAL ANNALS
of the District of Columbia

October 1968

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Continuing Medical Education and Training in the Washington Metropolitan Region

LEONARD CHIAZZE, JR., Sc.D.,* AND THOMAS W. MATTINGLY, M.D., F.A.C.P.†

CONTINUING MEDICAL EDUCATION and training are considered to be important activities of the Regional Medical Program of Metropolitan Washington. The importance of these activities for Regional Medical Programs has been well recognized both by the staff of the Division of Regional Medical Programs, PHS and Regional Programs throughout the country. Consequently, one of the initial activities of the Washington Metropolitan Regional Program was to conduct a survey of continuing medical education of physicians in the area. The survey had the following objectives:

1. Obtain a summary of the present status of continuing education and training in the region.
2. Develop information regarding continuing education and training over the past several years.
3. Identify existing patterns of continuing education for the region.
4. Determine the techniques preferred by physicians in the region.
5. Identify factors which inhibit the effectiveness of present programs.
6. Identify those physicians or groups of physicians who do not utilize current programs for continuing education.
7. Identify special areas in which deficiencies exist and which might be strengthened through programs of the Regional Medical Program.

In short, it was our objective to identify unfulfilled needs for and barriers to continuing medical education in the region so that plans for future programs could be made that would reach the widest possible audience.

Materials and Methods

At the time of this survey, in early 1967, there were 4,124 physicians registered in the AMA files for the city of Washington, D. C. and the suburban counties of Maryland and Virginia which comprise the Washington Metropolitan Region. Excluded from this number are interns, residents, fellows, trainees, physicians commissioned in the 4 Federal Services, and those retired or not in active practice. However, we included those physicians in the various branches of the Federal Services who were serving in a civilian status and those who were full-time members of faculties of the medical schools and staffs of hospitals and medical institutions. Prior to the mailing of the survey questionnaire to each of the 4,124 physicians in the region, 2 editorials were published in the MEDICAL ANNALS OF THE DISTRICT OF COLUMBIA to alert practicing physicians to the pending survey. After distribution of the questionnaires, follow-up was attempted for those physicians who did not respond, first by a follow-up letter, and then by a personal phone call to nonresponding physicians. In addition, notices were placed in the *Newsletter* of the Medical Society and in the MEDICAL ANNALS urging physicians to complete the questionnaire and return it to the Regional Medical Program office.

Of the 4,124 physicians contacted, 3,374 were involved in direct patient care and 750 were not. The distribution for selected specialties is shown in table 1. For this and the remaining tables in this paper, 2 summary categories, i.e., Other Medical and Other Surgical, have been established by combining specialties as shown in the footnote in table 1. Thirty-two per cent of all physicians and 31 per cent of those involved in direct care of patients returned completed questionnaires. In general, the proportion responding by specialty did not vary greatly. For example, internal medicine had the highest per cent responding (39.3) and Other Surgical the lowest (23.6). The proportion responding was greater than 30 per cent for all but the surgical specialties. For most specialties, the per cent responding is about the same for physicians engaged in direct care of patients as for all physicians in that specialty.

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† Program Coördinator, Metropolitan Washington Regional Medical Program, 2007 Eye Street, N.W., Washington, D. C. 20006.

TABLE 1: Total Number of Physicians Included in Survey of Continuing Education and Per cent Responding by Specialty

	All Physicians		Physicians in Direct Care of Patients		Phyicians Not in Direct Care of Patients	
	Total Number	Per cent Responding	Total Number	Per cent Responding	Total Number	Per cent Responding
All Physicians	4,124	31.8	3,374	31.1	750	36.3
General practice	654	31.5	648	31.6	6	33.3
General surgery	276	28.6	243	28.4	33	27.3
Internal medicine	659	39.3	553	39.4	106	40.6
Other Medical specialties*	169	37.9	137	35.0	32	50.0
Other Surgical specialties†	449	23.6	429	23.8	20	15.0
Obstetrics and gynecology	304	32.2	284	31.7	20	40.0
Pediatrics	297	32.0	257	32.7	40	27.5
Administrative medicine	246	35.4	0	—	246	35.4
Pathology	158	32.9	58	25.9	100	37.0
Psychiatry	439	32.6	404	31.7	35	42.9
All other	473	27.5	361	24.7	112	36.6

*Includes allergy, cardiovascular disease, dermatology, gastroenterology, neurology, and occupational medicine.

†Includes neurological surgery, ophthalmology, orthopedic surgery, otolaryngology, plastic surgery, colon and rectal surgery, thoracic surgery, and urology.

Clearly, we have no way of determining how representative the respondents may be of the total number of physicians in each specialty. The sample may very well be biased, and any inferences which are attempted from the approximately one-third of the physicians who did respond must be made with great caution. On the other hand, the data can be used to provide minimal estimates for participation in continuing medical education as measured by the parameters used herein. One further caution is in order. The data in this paper are physician-oriented and in no way provide a measure of benefits which may accrue to patients as a result of efforts at continuing medical education on the part of the physicians.

Results

A distribution by specialty of the number of physicians with hospital and medical school faculty appointments during the past 10 years is shown in table 2. The specialties listed make up 89 per cent of all responding physicians. Nearly 11 per cent of all responding physicians have no hospital appointment of any kind, although there is considerable variation by specialty. About half of the responding physicians with no hospital appointment are in the specialties of administrative medicine, pathology, and psychiatry. More important may be the observation that 9 per cent of the general practitioners had no hospital appointment, while only 3 per cent of those in the specialties of general surgery and internal medicine had no hospital appointment. While it may be surprising that nearly 16 per cent of those in Other Medical had no appointment, the data may be reconciled by the following. Nineteen per cent of those in this specialty are not involved in patient care and half of these responded to the survey. This would serve to inflate the proportion having no hospital staff appointment. In addition, the numbers are quite small and subject to a fair degree of variability. Consequently, the 16 per cent figure is not as far out of line as it seems.

TABLE 2: Hospital and Medical School Faculty Appointments by Specialty

	Total Number	Hospital Appointments		Medical School Faculty Appointments	
		None (Per cent)	Active or Other (Per cent)	None (Per cent)	One or More (Per cent)
All Physicians	1,310	10.5	89.5	43.7	56.3
General practice	206	9.2	90.8	89.3	10.7
General surgery	79	2.5	97.5	46.8	53.2
Internal medicine	259	3.5	96.5	25.9	74.1
Other Medical	64	15.6	84.4	21.9	78.1
Other Surgical	106	2.8	97.2	25.5	74.5
Obstetrics and gynecology	98	4.1	95.9	43.9	56.1
Pediatrics	92	4.3	95.7	34.8	65.2
Administrative medicine	84	41.7	58.3	51.2	48.8
Pathology	54	18.5	81.5	44.4	55.6
Psychiatry	141	16.3	83.7	36.9	63.1
All other	127	14.2	85.8	38.6	61.4

There is much greater disparity among the specialties with no medical school faculty appointment. The proportion of all respondents with no appointment was 44 per cent, while 89 per cent of the general practitioners as compared with only 26 per cent of those in internal medicine had no appointment of this type. The disparity is extremely worth noting and can provide some potential insight into future action for the Regional Medical Program.

TABLE 3: Per cent Distribution of Related Professional Activity Engaged in During the Past Year and Five Years Ago by Specialty

Type of Professional Activity and Specialty	Frequency of Attendance									
	Past Year					5 Years Ago				
	None (%)	Weekly (%)	Monthly (%)	7–11/Year (%)	1–6/Year (%)	None (%)	Weekly (%)	Monthly (%)	7–11/Year (%)	1–6/Year (%)
Hospital Staff Conferences										
All Physicians	15.5	37.5	19.3	15.7	11.9	21.9	36.3	18.5	12.4	10.9
General practice	14.6	18.0	26.2	22.8	18.4	17.5	17.0	27.2	19.4	19.0
General surgery	—	62.0	16.5	12.7	8.9	13.9	53.2	19.0	10.1	3.8
Internal medicine	6.6	61.4	13.9	12.4	5.8	15.4	53.3	14.7	10.8	5.8
Other Medical	18.8	42.2	17.2	9.4	12.5	26.6	42.2	12.5	6.3	12.5
Other Surgical	5.7	39.6	27.4	22.6	4.7	23.6	34.0	21.7	16.0	4.7
Obstetrics and gynecology	5.1	23.5	41.8	24.5	5.1	13.3	25.5	36.7	19.4	5.1
Pediatrics	9.8	33.7	27.2	14.1	15.2	21.7	28.3	23.9	15.2	10.9
Administrative medicine	47.6	14.3	3.6	9.5	25.0	35.7	32.1	7.1	7.1	17.8
Pathology	29.6	33.3	13.0	13.0	11.1	35.2	37.0	13.0	11.1	3.7
Psychiatry	30.0	29.3	15.0	10.0	15.7	31.2	34.8	12.8	7.1	14.2
All other	20.5	40.9	10.2	16.5	11.8	25.2	39.4	10.2	7.9	16.5
Social Professional, Medical & Specialty Societies:										
All Physicians	34.1	3.8	17.6	12.0	32.3	46.1	3.2	14.7	10.5	25.4
General practice	43.9	0.5	15.0	11.5	29.1	51.1	0.5	14.3	9.2	24.9
General surgery	16.9	5.9	34.2	10.5	30.8	37.1	6.3	24.6	9.3	20.3
Internal medicine	32.2	4.1	18.3	12.0	33.4	44.3	3.5	14.4	11.6	26.3
Other Medical	34.9	6.8	13.5	13.6	31.2	49.0	4.7	11.3	14.6	20.8
Other Surgical	17.7	6.3	30.2	15.7	30.2	39.3	4.7	20.4	11.3	24.2
Obstetrics and gynecology	23.8	2.7	18.0	16.0	39.5	35.7	3.0	17.4	12.9	30.9
Pediatrics	35.5	2.5	19.6	7.6	34.8	48.2	2.5	16.0	6.9	26.4
Administrative medicine	49.6	1.2	4.8	11.5	33.0	50.0	1.6	6.3	13.5	28.6
Pathology	47.6	5.6	13.6	8.1	25.3	59.3	3.7	11.1	6.2	19.7
Psychiatry	34.3	5.2	16.4	12.6	31.2	46.6	4.5	14.2	11.3	23.4
All other	37.8	4.7	12.1	11.0	34.4	49.6	3.4	10.2	7.6	28.3

One of the more important questions included in the survey referred to the type of related professional activity which physicians engaged in the year prior to the survey and 5 years earlier. This question included activities such as hospital staff conferences, attendance at social professional societies, medical society meetings, and specialty meetings such as those of the heart association, cancer society, tumor board, and others. The results of this inquiry are summarized in table 3. The most important point is that 16 per cent of all physicians responding did not attend a single hospital staff conference during the preceding year. Again, the proportion varies greatly by specialty. For example, there were no general surgeons who did not attend at least 1 hospital staff conference during the past year. Among general practitioners, the proportion not attending hospital staff conferences was 15 per cent, while 19 per cent of Other Medical specialties did not attend a single conference. The highest proportions for nonattendance were noted for the specialties of administrative medicine, psychiatry, and pathology. But this level of attendance is more in line with expectations than with the preceding specialties. It should be noted, however, that there has been a substantial improvement in attendance at hospital staff conferences and in other types of professional activities when the activities of the preceding year are compared with those of 5 years ago. For example, 5 years ago 22 per cent of the physicians responding did not attend a single staff conference and the proportion for general practitioners at that time was 18 per cent and for general surgeons was 14 per cent, so that, using this indicator, there has been improvement over the past 5 years.

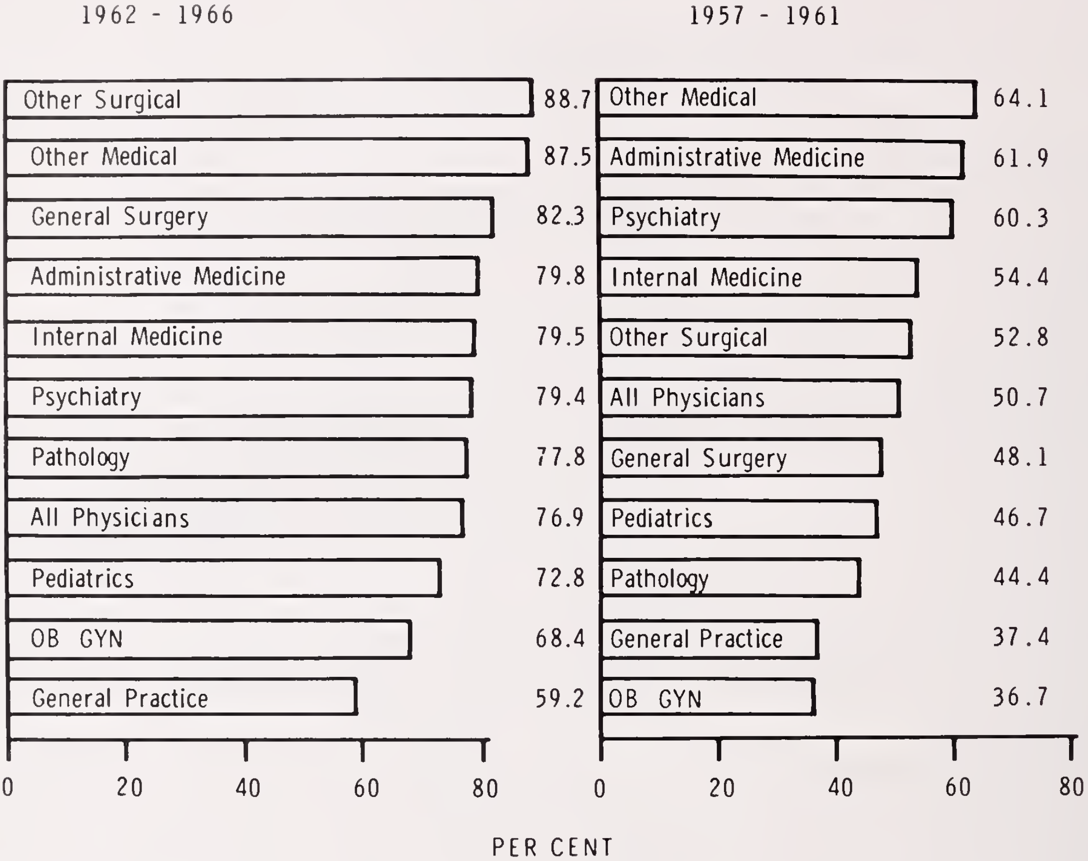

FIG. 1. Per cent distribution of attendance of 1 or more professional meetings in 1962–66 and 1957–61 in rank order by specialty.

Figures 1 and 2 provide some insight into 3 factors related to participation in continuing education. These factors are attendance at professional meetings, attendance at formal courses in medical education, and visits to a center for purposes of learning a new technique in diagnosis or therapy. The vast majority of physicians in all specialties attended at least 1 professional meeting during the past 5 years. Surgical specialists had the highest attendance, followed closely by medical specialists. General practitioners attended meetings less frequently than any of the specialists listed. A somewhat different picture is gained from the distribution of attendance at formal courses in medical education, where one sees that the medical specialists have the highest proportion in attending 1 or more meetings, while the general surgeons have the lowest percentage and general practitioners about twice that of general surgeons. Visiting a center for the purpose of learning a new technique seems to be far less popular than either attendance at professional meetings or attending formal courses. The percentage of all physicians who made 1 or more visits is on the order of 30 per cent. Again, the most active specialists for this particular method are the surgeons; least active would appear to be pediatricians, general practitioners, and obstetricians and gynecologists. However, for both professional meetings and formal courses there has been a substantial increase in attendance from the 1957–1961 period to the more recent 1962–1966 period.

Perhaps the most widely recognized way of communicating new information and keeping up to date is through the regularly distributed professional journals. Table 4 provides an indication of the distribution by specialty of the number of medical journals read regularly. Only 7 per cent of all responding physicians indicated that they read no journals on a regular basis. On the other hand, 46 per cent of the respondents said they read 5 or more journals regularly, although this varies considerably by specialty. For example, 52 per cent of the general surgeons indicated such a frequency as compared with 37 per cent of the general practitioners. However, only 6 per cent of the general practitioners indicated that they read no journals regularly as compared to over 10 per cent for those in general surgery, administrative medicine, and pathology.

TABLE 4. Per cent Distribution of Number of Medical Journals Read Regularly by Specialty

	None	One	Two	Three	Four	Five or more
All Physicians	6.9	3.4	9.3	16.6	17.5	46.3
General practice	5.8	5.3	13.1	18.4	20.4	36.9
General surgery	10.1	2.5	3.8	13.9	17.7	51.9
Internal medicine	5.0	1.5	8.5	16.6	18.9	49.4
Other Medical	4.7	3.1	1.6	10.9	10.9	68.8
Other Surgical	3.8	3.8	12.3	21.7	23.6	34.9
Obstetrics and gynecology	8.2	5.1	10.2	21.4	15.3	39.8
Pediatrics	4.3	6.5	10.9	20.0	12.0	46.7
Administrative medicine	10.7	—	16.7	15.5	10.7	46.4
Pathology	11.1	—	3.7	5.6	13.0	66.7
Psychiatry	9.2	12.7	8.5	17.7	22.0	36.2
All other	8.7	1.6	6.3	11.8	15.0	56.7

Discussion

The results of this survey would seem to suggest that, although there has been some improvement in continuing medical education on the part of the physician in the Washington Metropolitan Region over the past several years according to the parameters used, there remains substantial need for improvement. Advances in medical knowledge are growing at an unprecedented rate. It is one of the objectives of the Regional Medical Program to

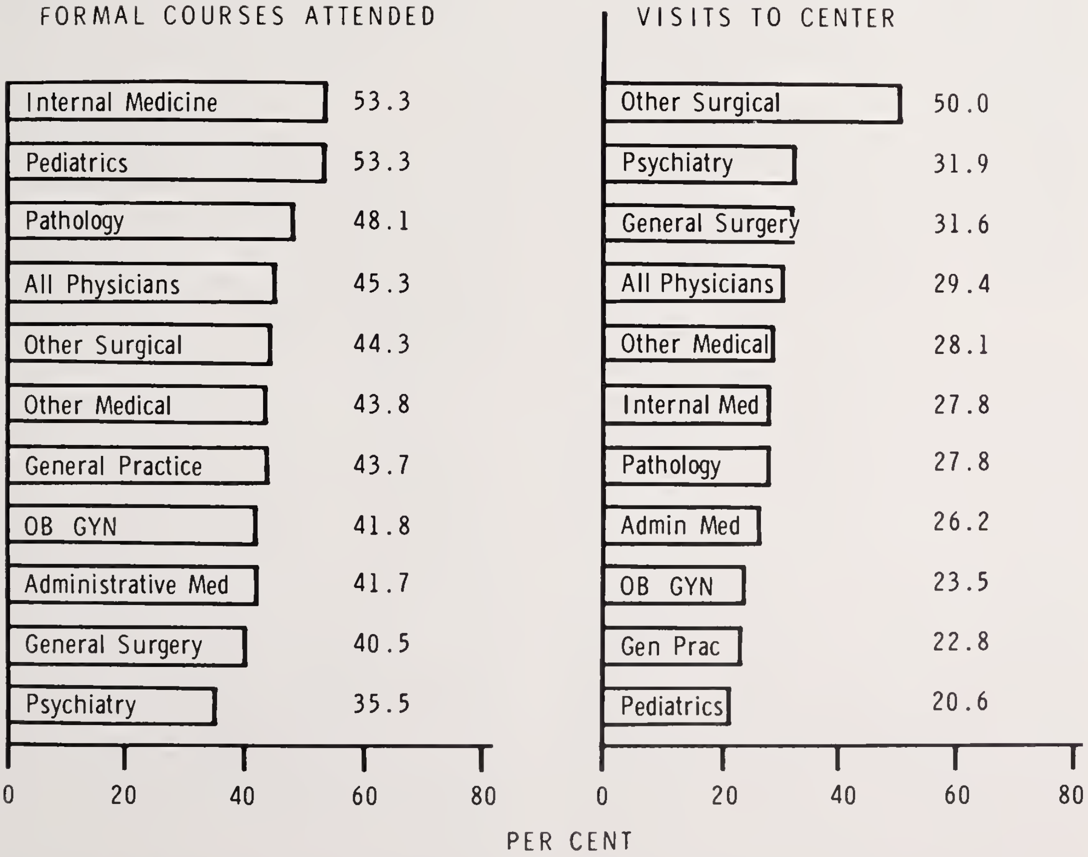

FIG. 2. Ranking of attendance (per cent) at 1 or more formal courses in medical education (1962–66) and 1 or more visits to center to learn new techniques by specialty.

bring these advances in medical knowledge to the patient in the quickest possible way through the physician. Clearly, the traditional methods for communicating information—the medical journal, the hospital staff conference, and the professional meetings—have been utilized to a fair degree by the responding physicians. On the other hand, the variation in response to available programs by specialty suggests that there should be some specialty-oriented programs with methods which will make it much easier for the physician to participate in and not take time away from his practice. For example, about three-quarters of the responding physicians found some type of audiovisual aid helpful in continuing education (table 5). Audiotapes and motion pictures would appear to be favored about equally by the majority of the specialists responding, followed by demonstration units and, bringing up the rear, the use of television. Even though television could be utilized to a much greater extent, motion pictures and audiotapes may provide a greater flexibility of scheduling for the physician. Perhaps it is in this area that one might want to concentrate efforts. When physicians were asked what types of programs they would like to see instituted, 21 per cent indicated that television would be valuable (table 6). The response most frequently indicated, however, was that of formal courses followed closely by hospital staff conferences. At least as far as respondents to this survey are concerned, the latter 2 methods appear to be those most desired in a program of continuing education. Each of these responses indicates that the medical school, and the teaching hospitals especially, can have an exceedingly great impact on continuing education by providing the kind of continuing education which the local physicians would find valuable. Clearly, the majority of the physicians in each of the specialties given in the table could be reached through the mechanisms of formal courses and hospital staff conferences. This notion is not necessarily negated by the fairly unexpected proportion who do not attend hospital conferences, for it must be admitted that there will be some physicians who have no interest in pursuing a continuing education and training on any basis. Clearly, merely talking about it is not going to reach these individuals.

TABLE 5: Per cent Distribution of Types of Audiovisual Aids Found Helpful in Continuing Education by Specialty

	None	Audio-tapes	Moving Pic-tures	TV	Demon-stration Units	Other
All Physicians	24.9	30.7	28.7	4.0	5.2	6.5
General practice	22.8	30.6	28.6	7.8	7.8	2.4
General surgery	12.7	29.1	44.3	2.5	3.8	7.6
Internal medicine	23.9	33.6	25.9	3.5	4.6	8.5
Other Medical	26.6	12.5	40.6	1.6	7.8	10.9
Other Surgical	16.0	21.7	48.1	3.8	3.8	6.6
Obstetrics and gynecology	20.4	46.9	20.4	5.1	3.1	4.1
Pediatrics	27.2	37.0	19.6	6.5	5.4	4.3
Administrative medicine	29.8	19.0	36.9	2.4	6.0	6.0
Pathology	33.3	20.4	20.4	3.7	7.4	14.8
Psychiatry	46.1	30.5	14.2	1.4	1.4	6.4
All other	15.7	37.8	29.9	3.1	7.1	6.3

TABLE 6: Per cent Distribution of Types of Local Programs Desired for Future Continuing Education by Specialty

	Formal Courses	Demonstra-tion Units	Television	Visiting Consultants	Hospital Staff Conferences	Teaching Rounds	Other
All Physicians	44.4	27.8	21.1	39.2	40.8	35.6	7.0
General practice	51.4	37.4	29.6	34.0	43.7	29.6	5.3
General surgery	35.4	22.8	35.4	54.4	54.4	54.4	6.3
Internal medicine	51.0	32.4	22.8	47.5	54.4	49.4	5.0
Other Medical	45.3	29.7	7.8	37.5	43.8	37.5	6.3
Other Surgical	39.6	19.8	17.9	50.9	39.6	39.6	7.5
Obstetrics and gynecology	53.1	32.7	30.6	48.0	39.8	32.7	1.0
Pediatrics	53.3	18.5	21.7	39.1	41.3	39.1	3.3
Administrative medicine	32.1	22.6	13.1	15.5	27.4	40.5	8.3
Pathology	50.0	35.2	18.5	35.2	24.1	14.8	5.6
Psychiatry	24.1	10.6	7.8	27.7	21.3	17.7	19.9
All other	43.3	33.9	18.1	36.2	37.0	26.0	7.1

TABLE 7: Per cent Distribution of Factors Affecting Participation in Continuing Education by Specialty

	No Difficulty	No Desire to Attend	Programs Distant or Not Satis-factory	Interfere with Time Committed to Patients	Combination of Factors	All Other
All Physicians	40.8	3.6	11.5	13.3	17.3	13.5
General practice	22.8	2.4	11.2	16.5	28.2	18.9
General surgery	50.6	—	11.4	11.4	13.9	12.7
Internal medicine	45.6	1.5	8.5	14.7	20.8	8.9
Other Medical	48.4	—	12.5	4.7	10.9	23.4
Other Surgical	64.2	—	8.5	10.4	8.5	8.5
Obstetrics and gynecology	33.8	2.0	11.2	22.4	20.4	10.2
Pediatrics	35.9	4.3	12.0	18.5	23.9	5.4
Administrative medicine	44.0	8.3	19.0	2.4	6.0	20.2
Pathology	44.4	9.3	7.4	9.3	13.0	16.7
Psychiatry	44.0	11.3	14.2	8.5	10.6	11.3
All other	32.3	3.1	14.2	16.5	15.0	18.9

(*Continued on page 542*)

Hemodynamic Cardiovascular Response to Raised Intracranial Pressure*

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AND COLONEL LUDWIG G. KEMPE, MC, USA¶

A PRESSOR RESPONSE in which the mean arterial blood pressure stabilizes at a level above that of raised intracranial pressure was first demonstrated in 1881 in Germany[1] and was later confirmed in 1901 in the United States by Cushing.[2] This cardiovascular response serves to maintain the circulation to the brain in diseased states like tumor, hematoma, etc. Several groups of investigators have further characterized parts of the hemodynamic changes, but there was no agreement as to the precise mechanism in which the arterial blood pressure is elevated.[3-9] We recently have reported clinical and experimental studies concerned with increased intracranial pressure and pulmonary edema wherein we defined the role of the pulmonary hemodynamics of the Cushing reflex.[10, 11] These studies were expanded to include cardiac output, central blood volume, and total peripheral resistance in large experimental primates, chimpanzees, whose blood volume would not be altered significantly by repeatedly performing these latter studies. Because of the seeming conflicts in the literature we elevated intracranial pressure stepwise in order to define the exact sequence of cardiovascular events wherein cerebral profusion is maintained.

Methods and Materials

The principles of laboratory care as promulgated by the National Society for Medical Research were observed. Adult chimpanzees of both sexes were sedated with 40 mg. of phencyclidine hydrochloride administered intramuscularly by a tranquilizer pistol and were anesthetized lightly with intravenous sodium pentobarbital (20 mg./Kg). Ventilation with room air was controlled by a Harvard constant volume ventilator at a rate and volume sufficient to maintain normal arterial blood pO_2, pCO_2, and pH. Polyethylene catheters for monitoring pressures were placed in the abdominal aorta, right atrium, and pulmonary artery via the femoral or jugular vessels. Catheters were placed in the left atrium at thoracotomy. All pressure recordings were made with calibrated transducers connected to appropriate pre-amplifiers and an amplified recording system (Sanborn 150 Recorder). The zero reference level for all pressures was one-third the anterior-posterior thickness of the thorax behind the anterior sternal surface. Cardiac output was determined by the indicator dilution method with the use of indo-cyanine green. The mean transit time was computed from the time-concentration curve. From these data the central blood volume (pulmonary outflow tract → abdominal aorta) and total peripheral resistance were calculated. The heart rate was determined from electrocardiographic tracings. Intracranial pressures were recorded via a polyethylene catheter placed in the floor of the middle cranial fossa through a burr hole over the parietal lobe. An epidural balloon was placed intracranially with a metal shield holding the intracranial apparatus in place. The balloon was inflated sufficiently to produce the desired intracranial pressure. The balloon volume never exceeded 35 c.c. In order to maintain a constant intracranial pressure, small increments of saline had to be added to the balloon; this apparently represents a degree of shrinkage of the intracranial contents in response to the increased pressure.[12]

All animals were killed by a large dose of intravenous pentobarbital at the end of the experiment. Autopsies were performed to study

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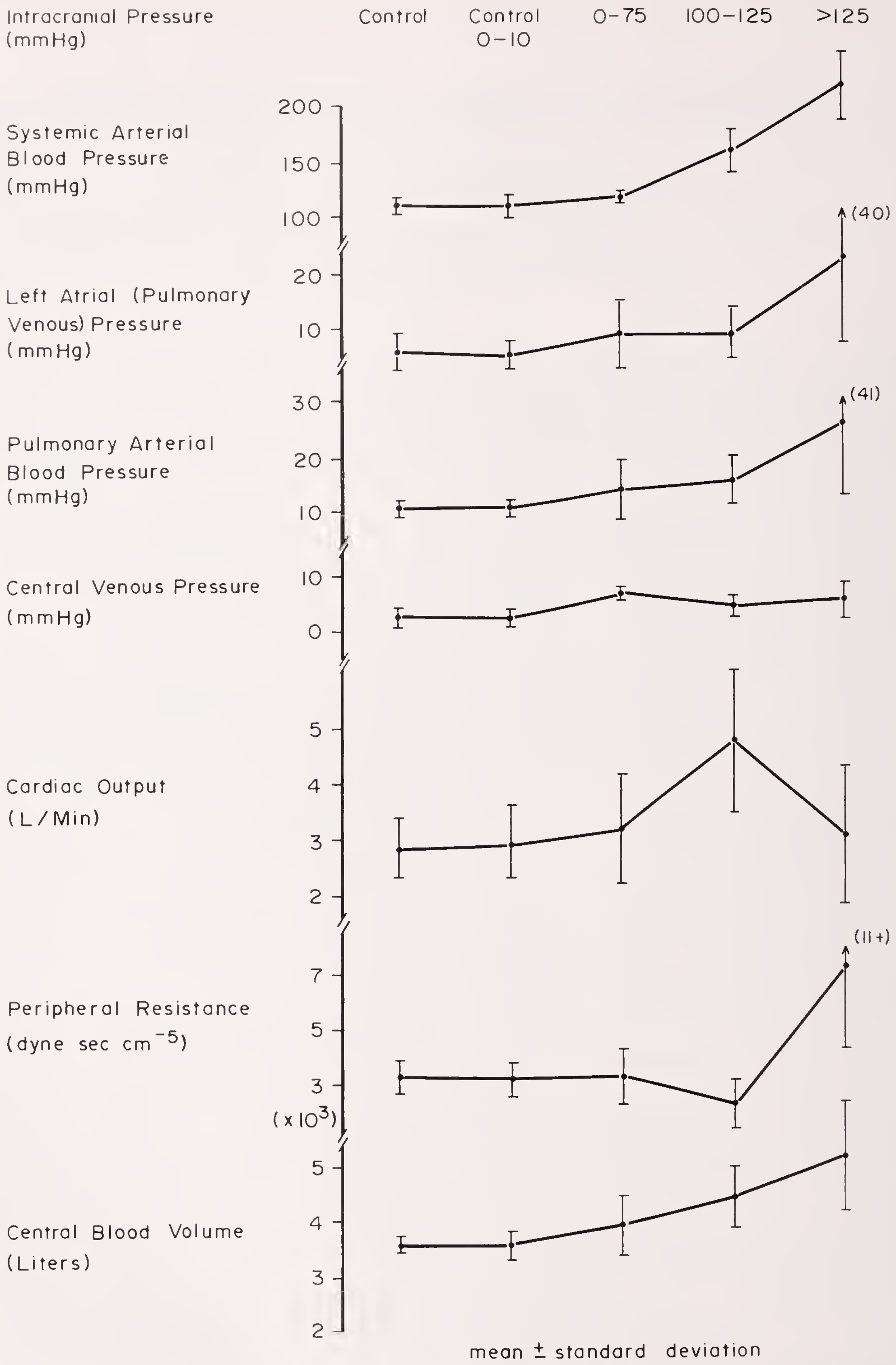

FIG. 1. Hemodynamic cardiovascular recordings in the control state, the second control state with intracranial balloon and catheters in place, and under increased intracranial pressure. The pressures recorded are the mean. The limits of all determinations are the standard deviations.

the cerebral damage and the changes in the lungs. Tissues were fixed in buffered formalin, embedded in paraffin, and sectioned at 6 μ. Hematoxylin and eosin staining was routinely performed.

Results

The results from 7 chimpanzees are summarized in figure 1; with intracranial pressures up to 75 mm. Hg, there was no consistent significant cardiovascular alteration except for an increase in the central venous pressure (probability value <0.01). No immediate change was detected in cardiac output or total peripheral resistance. At 100 mm. Hg the systemic arterial pressure and cardiac output rose ($p < 0.01$). The occasional presence of a transient slowing of heart rate did not alter the consistent finding of an increased cardiac output. The central blood volume rose, and the total peripheral resistance decreased ($p < 0.01$). When the increased intracranial pressure was raised above 125 mm. Hg the systemic arterial pressure rose to a level to the point that diastolic pressure exceeded intracranial pressure. By the time systemic arterial blood pressure had stabilized the total peripheral resistance had reached extremely high levels ($p < 0.01$) and the cardiac output had returned toward normal levels. These changes were accompanied by gradual increases in pulmonary arterial and left atrial pressures and central blood volume. Two of the animals manifested total peripheral resistance levels greater than 10,000 dyne sec. cm.$^{-5}$, and pulmonary edema ensued.[13] At autopsy the displacement of the intracranial contents by the epidural balloon was obvious in all 7 animals. Small brain-stem hemorrhages were seen in 2 animals. Evidence of a tentorial pressure cone was present in all. The microscopic appearance of the brain in these acute experiments was otherwise not remarkable. The lungs of the 2 animals who developed left-sided heart failure showed pulmonary edema, while the lungs of the other 5 were normal. The hearts of all animals appeared normal, and there was no valvular disruption in the animals with pulmonary edema.

Discussion

This cardiovascular hemodynamic response is so consistent that it would seem to represent a mechanism by which the organism may maintain brain perfusion despite the pressure exerted on the brain. It is unclear whether the stimulus for this response originates in the cerebral ischemia or in the intracranial pressure itself. However, once a stimulus has activated the hypothalamic, medullary, and spinal cord pressure centers, the nervous system appears to be integrated in such a way to maintain systemic arterial pressure above intracranial pressure.

When intracranial pressure was raised to 75 mm. Hg, only the first step of the reflex is called into play with an increase in central venous pressure which most probably represents mobilization of the intravascular volume with venoconstriction.

At 100 mm. Hg there is an increase in cardiac output appearing concomitantly with the initial arterial pressor response. These changes are preceded by a slight increase in central venous pressure and central blood volume and are never associated with increases in total peripheral resistance. The pressor response in these cases apparently results from an increase in venous return and/or a primary inotropic effect on the heart itself. Brown[3] has shown that there is an increase in venous tone following transient increases in intracranial pressure, presumably due to increases in sympathetic discharge or increases in circulating catecholamines. The resulting increase in venous return could account for the increased cardiac output. Sarnoff[14] also demonstrated that stimulation of sympathetic nerves to the heart may result in transient reversible increases in cardiac contractility. This effect also may help to account for the increased cardiac output.

As the intracranial pressure is further increased above 125 mm. Hg the cardiac output remains elevated. At this point the systemic blood pressure rise is accompanied by increases in the total peripheral resistance. Such marked increases of peripheral resistance may enforce a sudden work load on the heart leading to a moderate fall in cardiac output (figure 1). If peripheral resistance is reflexly raised to extremely high levels ($>10{,}000$ dyne sec. cm.$^{-5}$), the strain on the left ventricle becomes excessive, the left atrium distends, left atrial (pulmonary venous) pressure rises quickly and markedly, and pulmonary edema ensues.

These experiments strongly indicate that gradations of autonomic discharge are responsible for the pressure changes observed in the laboratory and in clinical practice. The data also give insight into the clinical complications in the cardiovascular system.

Summary

A number of previous investigators[3, 4] have implicated increased venous tone and return, a

(*Continued on page 574*)

Some Effects of Hydralazine on Hypertensive Patients with Impaired Kidney Function[*]

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GLOMERULAR FILTRATION RATE (C_{In}) and renal plasma flow (C_{PAH}) are reduced in patients with impaired kidney function.[1] Antihypertensive drugs which lower cardiac output and renal blood flow may, therefore, be less suitable in the treatment of patients with impaired kidney function than hydralazine which has been reported to reduce blood pressure while increasing cardiac output[2] and renal plasma flow.[3]

Reubi[4] compared the effect of a 4-week treatment with chlorothiazide, ganglionic blocking agents, reserpine, and hydralazine, and found that hydralazine was the most suitable drug as far as renal function was concerned (C_{In} + 3.9 per cent/C_{PAH} −0.5 per cent). However, he observed considerable individual differences in response: C_{In} +20 per cent/−19 per cent, C_{PAH} +8 per cent/−13 per cent.

The purpose of this study was to investigate hypertensive patients with impaired renal function in order to determine what parameters other than reduction in blood pressure are affected by hydralazine treatment and how they may influence renal function.

Methods

Patients with a diastolic blood pressure ≥ 110 mm. Hg and signs of impaired renal function (BUN $\geq$ 21 mg. and/or glomerular filtration rate ≤ 75 ml./min.) but without a history of cerebral vascular accident, angina pectoris, or electrocardiographic evidence of previous infarction were selected. Of the 11 patients in this study 1 was a known diabetic controlled on insulin. Of the remaining 10 patients glucose tolerance tests showed 7 to have a diabetic type of curve, but none required other than dietary treatment.

Before hospital admission the patients had a physical examination, chest and skull X-ray films, intravenous pyelograms, urinalysis, urine culture, an estimation of catecholamines, 17 hydroxy- and 17 ketosteroids, protein-bound iodine, and complete blood count to exclude any treatable cause of hypertension. The 24-hour creatinine clearance ($C_{Creat.}$) was determined.

Patients were admitted after they were off antihypertensive treatment for at least a week. Patients who received digitalis were kept on the same dose throughout the study. All patients received 5 Gm. salt and 40 Gm. protein in a 2,000-calorie diet. Estimation of alkaline phosphatase, cholesterol, serum bilirubin, SGOT, SGPT, serum electrophoresis, glucose tolerance test with insulin estimation,[5] urea nitrogen, uric acid, electrolytes, complete blood count, reticulocytes, sickle-cell preparation, erythrocyte sedimentation rate, clotting time, platelet count, lupus erythematosus preparation, and fundoscopic photographs were done to establish a baseline.

Two days after admission a renal function test was performed. One hour after 1,000 ml. fluid intake, 50 mg./Kg inulin and 8 mg./Kg PAH were injected intravenously as a priming dose, and an additional 500 ml. of water was administered by mouth. The blood levels of PAH and inulin were kept near constant by a sustaining infusion. A half hour after the priming dose the patients voided and a 2-hour clearance period was started. The fluid loss by voided urine was replaced by oral administration of an equal amount of water. After a 2-hour control clearance 20 mg. hydralazine was injected I.V. and the clearance study continued for another 2 hours. Blood pressure and pulse rate were checked every 10 minutes throughout the procedure. The mean blood pressure was calculated as the diastolic pressure plus one-third of the pulse pressure.

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After the renal function test, oral hydralazine treatment was started on a dosage increasing from 25 to 100 mg. 4 times daily according to the effect on the blood pressure. After a day's treatment a 24-hour creatinine clearance was done.

Patients were discharged, after the blood pressure was controlled, on a dose of hydralazine of 100 mg. 4 times daily if this could be tolerated without serious side-effects. Higher doses were not administered because of the rheumatoid arthritis and lupus erythematosus-like syndrome reported in the literature. Patients were seen at monthly intervals in the outpatient department. A questionnaire was filled out to ascertain side-effects. The blood pressure was checked after the patient had been lying down for 5 minutes and then after standing for 5 minutes. Twenty-four-hour creatinine clearance, blood urea nitrogen, uric acid, and electrolytes were estimated.

After 3 months of hydralazine treatment patients were readmitted for identical inpatient investigations but continued their hydralazine treatment with 4 daily doses. The morning dose was administered 1 hour before the renal function test was started.

In this way each patient served as his or her own control in the assessment of the acute and chronic effects of hydralazine.

All blood and urine estimations were carried out by accepted routine hospital methods. Inulin and PAH were determined by a semi-automated method as described by Krees et al.[6]

Results

After I.V. injection of 20 mg. of hydralazine the mean of the mean arterial blood pressure was reduced by 18.5 mm. Hg (S.D. ± 12.1, $P = 0.01$). At the same time the pulse rate increased by a mean of 13.8/min. (S.D. ± 8.9, $P = 0.01$). After the hydralazine injection the mean urine flow was reduced by 1.77 ml./min. (S.D. ± 2.39, $P = 0.05$). Concomitant with the decrease in urine flow there was a reduction ($P = 0.01$) in free-water clearance. As far as inulin- and PAH-clearance were concerned, there was no statistically significant change for the group, but some individuals showed clinically significant variations in either a positive or negative direction.

One hour after hydralazine injection 3 patients fainted when standing up to pass urine. Three other patients, who had also shown the highest drop in blood pressure after hydralazine injection, showed the highest percentage of decrease in urine flow.

Three patients complained of headaches and palpitation after hydralazine injection. These symptoms disappeared after a few hours although treatment was continued with hydralazine tablets.

During the outpatient treatment period when hydralazine was switched over from intravenous to oral application, the favorable effect on blood pressure reduction continued and was highly significant ($P = 0.01$), as compared to pretreatment levels. The 24-hour creatinine clearance increased at the same level of significance. The urine collection for this estimation was started 1 day after treatment had commenced.

The initial blood pressure reduction was observed to be poorly maintained after 1 month from discharge. The previously seen increase in 24-hour creatinine clearance was also less marked in most patients, and in 1 patient it was significantly reduced. With increased dosage of hydralazine it was possible to reproduce highly significant blood pressure reduction by the second month. However, in contrast, the 24-hour creatinine clearance did not improve, and after 3 months of treatment only 2 patients showed improvement of more than 20 per cent, while 2 other patients showed a significant deterioration. By the third month of treatment the blood pressure of the patients was no longer significantly reduced.

During hydralazine treatment only a few patients had some minor complaints like dizziness when getting up suddenly, light-headedness, palpitation, periorbital edema, and, in a severe case of hypertension, drowsiness. One patient developed marked tachycardia with premature atrial and nodal contractions and aberrant ventricular responses. His chest X-ray film showed minimal congestion in the peripheral pulmonary vessels. All these features disappeared when hydrochlorothiazide and reserpine were added to the therapy. In one patient who was on digitalis because of left ventricular failure they disappeared. However, after 2 months of treatment with hydralazine, her BUN increased from 28 to 82 mg. and she felt nauseated, vomited, and developed angina. When hydralazine treatment was stopped these symptoms disappeared and her BUN dropped slightly. In contradistinction the BUN of one patient increased from 16 mg. to 71 mg. when she was treated with hydrochlorothiazide, but it returned to normal again when treatment was stopped. The BUN remained in the normal range during treatment with hydralazine, although her blood pressure was only slightly less reduced than with hydrochlorothiazide.

When patients were readmitted after 3 months of treatment with hydralazine their blood pressure readings were significantly decreased (P = 0.05) as compared to inpatient pretreatment values. However, the patients as a group showed no significant changes in pulse rate, urine flow, glomerular filtration rate, renal plasma flow, and free-water clearance, although there were clinically significant individual changes, both increases and decreases in kidney function. C_{In} and C_{PAH} were strikingly reduced in those patients whose clinical examinations suggested progressive arteriosclerosis due to age and/or diabetes mellitus. Besides T-wave depressions in 2 patients with angina and tachycardia respectively, there were no electrocardiographic changes or alterations in the chest X-ray film, the fundi, oral glucose tolerance test, or other laboratory tests. We found no signs of rheumatoid arthritis or lupus erythematosus.

When during the second hospital admission 20 mg. of hydralazine was injected 2 hours after the beginning of the clearance test, the pulse rate of the patients increased significantly while alterations in blood pressure and kidney function remained without significance. The patients who had had the most striking reduction in renal plasma flow in connection with a drop of mean arterial blood pressure of 23 mm. Hg during the first admission reacted this time with a considerable increase in renal blood flow and no change in blood pressure.

Discussion

Our data demonstrate that the I.V. injection of 20 mg. of hydralazine reduced the blood pressure significantly (P = 0.01) without a significant change in glomerular filtration rate and renal plasma flow. However, urine flow and free-water clearance were reduced (P = 0.05 resp. 0.01). While this is true for the whole group, there are considerable individual differences in response. The antihypertensive effects of the 20 mg. dose were not inversely related to the body weight of the patients and showed no relation to the height of their blood pressure in the acute test.

Ranking the patients according to their drop in blood pressure showed that the 8 mild diabetic patients had a greater response than the 3 nondiabetic patients. Three of the diabetic patients with the highest reduction in blood pressure fainted when getting up to pass urine 1 hour after the hydralazine injection. They also showed the highest percentage in reduction of urine flow, glomerular filtration rate, and renal plasma flow. In view of the small number of patients in this study it is not possible to draw conclusions from these observations.

An abrupt reduction in blood pressure seems more likely to be the cause for decrease in urine flow rather than an effect of hydralazine itself, because one patient showed a 23 mm. Hg fall in his blood pressure and a more than 60 per cent fall in urine flow, glomerular filtration rate, and renal plasma flow when hydralazine was injected for the first time. After 3 months' treatment when the same dose was less effective and dropped his blood pressure by 3 mm. Hg his renal plasma flow increased, while all other kidney parameters remained unchanged. These data are in agreement with the findings of Reubi,[3] who saw an increase in heart rate and renal plasma flow at a 10 mg. dose, when the blood pressure was not consistently reduced in his patients. Judson et al[7] found a decrease in sodium and water excretion when hydralazine injection was followed by circulatory collapse, but, according to Hollander,[8] hydralazine had a rather diuretic effect, particularly in patients with congestive heart failure.

Our data confirm the diuretic effect in patients with congestive heart failure, and we saw oliguria in those who collapsed after hydralazine injection. However, in general the urine flow after hydralazine was decreased. The high percentage of diabetic patients in our group does not seem to be fortuitous. Rogers et al[9] reported a high incidence of azotemia in hypertensive patients with diabetic glomerulosclerosis.

Experiments with rats in our own laboratory* showed that in acute experiments over 5 hours, following a water load the urine flow increased up to a hydralazine dose of 6.75 mg./Kg, but urine flow decreased when still higher doses were administered, which presumably reduced the blood pressure considerably.

Controls	0.3 mg./Kg.	0.75 mg./ Kg.	1.5 mg./ Kg.	Doses of hydralazine per Kg., 4 animals per dose
10.4	18.8	28.5	19.7	ml. urine for 5 hrs.

During the first days of oral hydralazine treatment we observed a highly significant drop in blood pressure and at the same time a highly significant increase in 24-hour creatinine clearance. This may be in part due to the increased cardiac output induced by hydralazine[2] and in part to a reduction of renal arteriolar resistance. The reduction of central arterial pressure appears to be compensated by a decrease in renal afferent arteriolar resistance with maintenance of glomerular filtration pressure and consequent glomerular filtration rate. In general the positive effect of hydralazine on 24-hour creatinine clearance became less during the 3 months of treatment. Our data also show that changes in blood pressure and 24-hour creatinine clearance were independent; e.g., the decrease in blood pressure was highly significant after 2 months of treatment although there were no significant changes in 24-hour creatinine clearance as compared to pretreatment values.

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* To be described in detail elsewhere.

There is general agreement that malignant hypertension should be treated with antihypertensive drugs.[10, 11, 12] In nonmalignant hypertensive disease Moyer et al[13] compared untreated patients with those on antihypertensive therapy (ganglionic blocking agents and diuretics). He found a significant decrease in renal function in the untreated group, while glomerular filtration rate and renal plasma flow remained nearly unchanged in the treated patients. Differences between treated and untreated patients were more obvious when the diastolic blood pressure was higher than 130 mm. Hg.

There is no general agreement if and to what extent one may reduce the blood pressure in moderately azotemic hypertensive patients without further impairing renal function, and what drug is most suitable. Most popular antihypertensive drugs lower renal function because of their adverse hemodynamic effects. There are some favorable reports on antihypertensive treatment of azotemic patients with methyldopa.[14, 15] A disadvantage of this drug, however, is the decreasing effectiveness after a few months of treatment in most patients.[16]

Conclusions

Intravenous injection of 20 mg. of hydralazine reduced the blood pressure of 11 mild to severe hypertensive patients with impaired renal function in general without significantly changing glomerular filtration rate and renal plasma flow.

The response of the patients to a single injection of hydralazine was different. Those who reacted with a fall in mean arterial blood pressure of about 25 mm. Hg or more showed impaired renal function. Apart from hypertensive emergencies it seems, therefore, advisable to start with small doses of hydralazine and to adjust the dose according to the response of the patients.

Oral hydralazine is able to reduce the blood pressure for months and at the same time increase the glomerular filtration rate as measured by 24-hour creatinine clearance.

Hydralazine may reduce the blood pressure without impairing renal function in some patients who showed striking blood urea nitrogen elevations when on other antihypertensive drugs, e.g., diuretics.

Increasing cardiac output, even though patients may be already on digitalis, is a favorable effect of hydralazine and may lead, as our results show, to improved renal function. Tachycardia may be avoided or treated in some of these patients by adding reserpine. In cases of angina or unfavorable electrocardiographic changes hydralazine treatment should be stopped.

The effectiveness of hydralazine usually decreases after a few months of treatment, and as doses higher than 200–400 mg. daily and long-term use increase the risk of lupus erythematosus-like syndromes, hydralazine should be administered to hypertensive patients with impaired kidney function in turn with other antihypertensive agents, e.g., methyldopa. Hopefully other drugs will become available, e.g., dopamine-like compounds,[17] which have a favorable effect on renal hemodynamics and may be a useful adjunct in the treatment of the azotemic hypertensive patient.

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Renal Vein Thrombosis and the Nephrotic Syndrome in an Adult

Report of a Case and Review of the Literature

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ESTIMATES OF RENAL VEIN THROMBOSIS as the etiology of the adult nephrotic syndrome are between 1 and 5 per cent.[1, 2] The literature indicates than renal vein thrombosis is not always followed by a nephrotic syndrome. Renal vein thrombosis was found in 17 out of 29,280 autopsies (0.6 per thousand) and of these 2 definitely and 2 possibly had the nephrotic syndrome.[3] Because of the rarity of renal-vein thrombosis as a cause of the nephrotic syndrome the following case is reported.

Report of Case

B. W., a 25-year-old Negro woman (Case no. 420898), was admitted to the Washington Hospital Center on May 20, 1966 because of right-sided back pain, proteinuria, edema, and shortness of breath. Her illness began in October 1965 when she developed right-sided chest and shoulder pain. X-ray examination of the chest at this time revealed bilateral pleural effusion and a right lower-lobe parenchymal infiltrate. The patient was treated with tetracycline. Another chest X-ray examination after 6 days revealed clearing of the parenchymal process, but residual fluid remained in the right costophrenic angle. On October 28, 1965 the patient experienced burning on urination and was given streptomycin and sulfisoxazole. She was not seen again until December 27, 1965, at which time she complained of edema. Urinalysis revealed a specific gravity of 1.021, 4 + albumin, and 35 red blood cells per high-power field. On January 6, 1966 she complained of left-sided costovertebral angle pain and was treated again with sulfisoxazole. On February 18, 1966 she complained of right flank pain.

Abnormal urines persisted with proteinuria and microscopic hematuria to the time of admission to the hospital. The edema persisted despite the use of diuretics. The patient did not adhere to a low-sodium diet. Several days prior to admission she developed right flank pain of a pleuritic character associated with marked shortness of breath.

She had an appendectomy 12 years prior to admission. A pilonidal sinus was removed 3 years prior to admission, and a benign breast cyst was removed 1 year prior to admission. The family history was noncontributory, as was a review of systems.

The patient denied a history suggestive of peripheral thrombophlebitis, trauma to the abdomen or back, pelvic inflammatory disease, or prior renal disease. There was no history of abnormal urine examinations in the past, of acute glomerulonephritis, or of disseminated lupus erythematosus. The patient had not been exposed to nephrotoxic medications or chemicals. She was not taking progestational birth-control pills.

Physical examination revealed an obese Negro female in no distress, with weight, 170 lbs.; blood pressure, 140/70 in the right arm, 150/80 in the left arm; pulse, 80 and regular; respiration, 16. There were bilateral dependent 1+ edema to the midtibia, a right rectus skin scar, and thickening of the left uterosacral ligament. A venous pattern was not discernible over the abdomen or flanks.

Chest X-ray films showed fluid in the right costophrenic angle.

Laboratory studies obtained were as follows: hemoglobin, 13.0 Gm.; hematocrit, 39.5 per cent; white blood cells, 8,719, with a normal differential. Urinalysis: specific gravity between 1.010 and 1.030, 200 to 300 mg. protein on random determination, a few red and white blood cells, hyaline, fatty and granular casts, oval fat bodies, and doubly refractile fat bodies. The blood urea nitrogen was 7 mg. and serum creatinine, 0.65 mg. Twenty-four hour creatinine clearance was 107 c.c./minute, uncorrected for the patient's surface area. Twenty-four hour protein excretion on 3 determinations was 4.2, 3.4, and 4.0 Gm. LE preparations were negative on 4 occasions. Serum cholesterol was 242 mg. Total serum protein was 4.70 Gm. and was separated by electrophoresis as follows:

Albumin	1.95 Gm.
Alpha-1 globulin	0.36 Gm.
Alpha-2 globulin	0.82 Gm.
Beta-2 globulin	0.75 Gm.
Gamma globulin	0.82 Gm.

A urine culture showed no growth. A normal flora was obtained from throat and sputum cultures. Sputum specimens for tuberculosis revealed no acid-fast organisms on smear or culture. A glucose tolerance test was normal. A cervical Pap smear was Class I. Protein-bound iodine was 5.3 micrograms, and an iodine^{131}

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uptake was 32.7 per cent in 24 hours. An intravenous pyelogram showed notching along the upper third of the right ureter (figure 1).

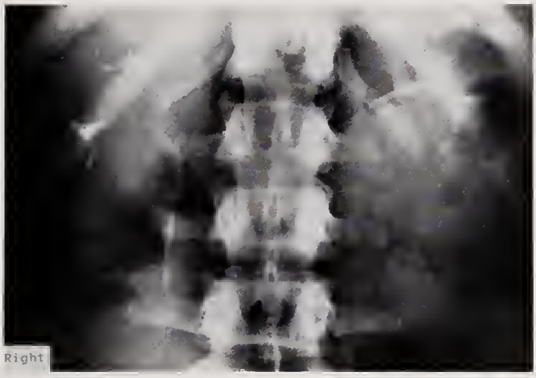

FIG. 1. Intravenous pyelogram showing notching along the upper one-third of the right ureter.

A percutaneous left renal biopsy was done. Sections of the tissue were described by Dr. Mary Jean Matthews, Chief, Section of Pathology, Veterans Administration Hospital, Washington, D. C.: "Sections revealed preservation of the usual renal architecture. Six glomeruli are present. The glomeruli have somewhat edematous capsules. Occasional epithelial cells lining Bowman's space are swollen. Bowman's spaces are prominent and empty. The basement membranes of the glomerular capillaries appear thick, sharp, and refractile in some areas and slightly thickened and edematous in others. There is focal proliferation and clumping of endothelial cells with obliteration of capillary lumina in these zones. Epithelial cells adjacent to these foci have swollen granular or vacuolated cytoplasm. Some capillaries are dilated, contain red blood cells, and others appear to contain a pale colorless fluid. Proximal convoluted tubules are lined by cells with moderately vacuolated or granular cytoplasm. There is moderate pleomorphism of nuclei. Distal and collecting tubules contain a small amount of protein precipitate. There is a minimal edema of the interstitial tissues and focal infiltration of lymphocytes and mononuclear cells. Arteries and arterioles are not remarkable. Veins are moderately dilated and filled with blood. With trichrome stains the basement membrane of the glomerular capillaries appears refractile, thick, and stains pale blue. With PAS-silver stains the basement membrane appears edematous and indistinct; occasional capillaries appear to have a thin argyrophilic substance outlining the inner and outer aspect of the basement membrane. Diagnosis: membranous and proliferative glomerulonephritis associated with renal vein thrombosis." (A representative glomerulus is shown in figure 2.)

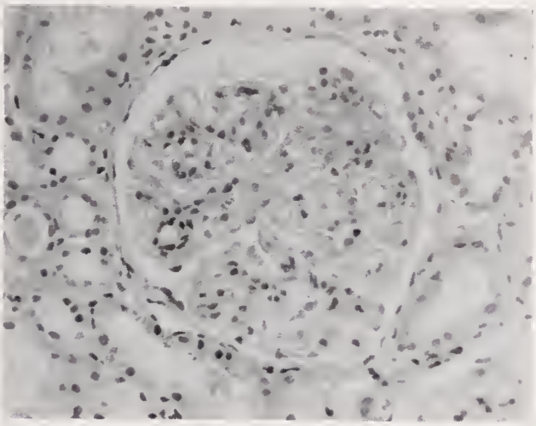

FIG. 2. Glomerulus showing basement-membrane thickening and a nonedematous interstitium.

Inferior vena cavography, an attempt at selective renal-vein catheterization, oxygen-saturation studies at various levels, and pressure recordings from the inferior vena cava at rest and with exercise were performed by Dr. James Bacos, Chief of the Department of Cardiology, Washington Hospital Center. The X-ray films showed a disturbed architectural pattern of the inferior vena cava at approximately the site of the renal-vein inflow. The inner wall of the inferior vena cava was ragged, suggesting thrombosis at this level (figure 3). Contrast material, however, seemed to flow readily into the right atrium. The renal veins were never opacified by injection at this point.

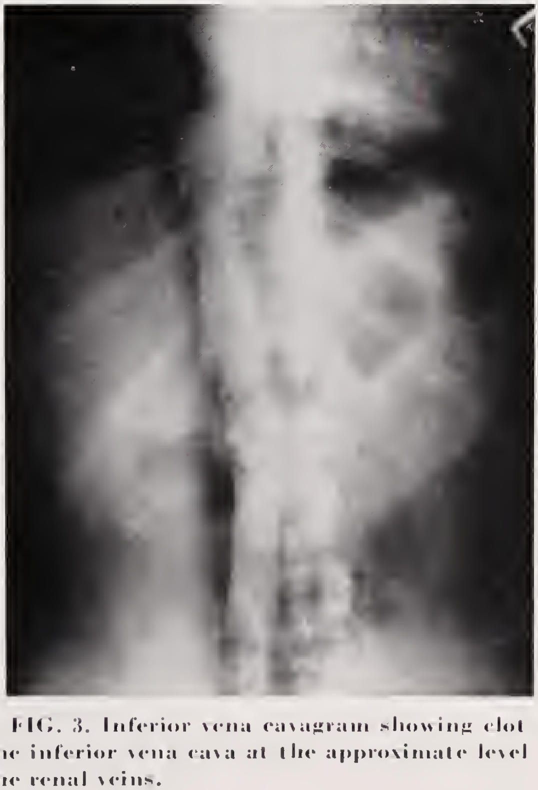

FIG. 3. Inferior vena cavagram showing clot in the inferior vena cava at the approximate level of the renal veins.

One week later another attempt was made to determine the patency of the renal veins. The right femoral vein was cannulated with a no. 8 Brockenbron catheter, and the catheter was passed up to the level of the renal veins. At this point, with the use of the Brockenbrou needle, the catheter was directed into what was thought to be the area of both right and left renal veins. At no time could the veins be cannulated; however, at the suspected points samples of blood were taken for oximetry. These were highly saturated at 86 and 88 per cent. Below this point the per cent of saturation dropped to 60. Repeatedly the catheter seemed to enter an area on the right at the approximate level where the right renal vein should enter. Injections at this point showed the contrast material to hang up but never in any definitive channel that resembled a right renal vein. Following attempts at introduction of the catheter into the renal veins a venous pressure obtained at rest was 18 mm. Hg. With exercise, the venous pressure at this level rose to 40 mm. Hg. These pressures were significantly elevated both at rest and with exercise and suggested some obstruction of the inferior vena cava flow.

The patient was treated with anticoagulant drugs and discharged on anticoagulants. Her status is unchanged as of June 1967.

Etiology of Renal Vein Thrombosis

Harrison[4] has summarized the pathogenetic mechanism of renal-vein thrombosis under 4 headings:

1. Thrombosis of the inferior vena cava with secondary involvement of the renal veins.
2. Obstruction of the inferior vena cava due to an invasion by malignant neoplasm or to external pressure.
3. Primary thrombosis of the renal veins.
4. Renal-vein thrombosis secondary to primary renal disease.

Inferior vena-caval thrombosis occurred in 11 out of 38 adults with renal vein thrombosis.[3] In turn, the inferior vena-caval thrombosis is frequently secondary to thrombosis in the leg or pelvic veins. The causes of inferior vena-caval thrombosis have recently been extensively reviewed.[5]

Anatomic studies in the inferior vena cava have revealed that thrombosis is most likely to occur in the inferior segment, that is, that area extending from the junction of the iliac veins to just below the renal veins.[6] It is this area where the flow of blood in the cava is slowest. Harrison[4] postulates that a sudden decrease in blood flow could enhance the extension of an inferior segment clot into the middle segment of the vena cava occupied by the renal veins.

Renal vein thrombosis associated with primary parenchymal renal disease has been attributed to such a decrease in renal blood flow.[7] The renal diseases associated with renal-vein thrombosis are numerous. The single renal disease that has been responsible for most recorded cases of renal vein thrombosis has been amyloidosis. Early case reports cite tuberculosis as the commonest cause of secondary amyloid and renal-vein thrombosis. A review of amyloidosis as an etiology of renal vein thrombosis in 1960 revealed 9 new and 39 old cases.[8] The newer cases were due to tuberculosis, perinephritis, ulcerative colitis, rheumatoid arthritis, and Hodgkin's disease. Dehydration, which was common in these patients with amyloidosis, was thought by the authors to be the precipitating factor leading to the renal-vein thrombosis. Although secondary amyloidosis is the most common type reported to cause renal-vein thrombosis, primary amyloidosis has been incriminated as well.[8,9]

Kowal et al[10] lists the predisposing diseases and factors leading to renal-vein thrombosis. These include such renal and urologic diseases as glomerulonephritis, pyelonephritis, ureterolithiasis, and hypernephroma. The nonrenal factors listed are trauma, thrombophlebitis migrans, metastatic carcinoma, malignant and essential hypertension, mercurial diuretics, sepsis, and urethral instrumentation. In addition, diabetic glomerulosclerosis[11] and polyarteritis nodosa[7, 12] have been incriminated as underlying causes. Congestive heart failure is conspicuous by its absence as an etiological factor in an earlier review of the literature.[13] Recent articles[3, 11] list congestive heart failure as the prime factor in renal-vein thrombosis in a high percentage of reported cases.

The finding of proliferative changes in the glomeruli in this patient does not correspond to the postmortem subacute glomerulonephritis described by Harrison (case 10).[4] She represents the third pathogenetic mechanism of renal-vein thrombosis—primary renal vein thrombosis.

Clinical Diagnosis

The age of reported cases of renal-vein thrombosis with the nephrotic syndrome ranges to 79 years. The average age is 47. Males predominate in a ratio of 2.5 to 1.[3] The most constant symptom is pain. It may be epigastric or occur in the flank or flanks.[3, 4, 7, 14] The pain may mimic the pain of renal colic. Pain was absent in more than half of a large reviewed series.[3] Renal-vein thrombosis may occur after abdominal surgery, and the pain may be masked by the postsurgical course.[15] Evidence by physical examination of a rapidly enlarging flank mass, the signs of peripheral thrombophlebitis, and evidence of pulmonary emboli and/or infarction reflect the intravascular thrombotic process.[14] The findings of hematuria[14] and later the typical lipid elements of the nephrotic syndrome[16] in the urinary sediment are further clues to the clinical diagnosis when taken in context with the evidence of intravascular thrombosis. Severe hypertension,[16] albeit rare, has been recorded as a manifestation of renal-vein thrombosis.

Edema of the lower extremities which precedes the onset of proteinuria is consistent with renal-vein thrombosis as the etiology of the nephrotic syndrome.[14] The temporal relationship of a peripheral thrombophlebitis and the renal-vein thrombosis may be distant.[13]

Thrombosis may occur unilaterally. In those cases with proven unilateral renal-vein thrombosis, proteinuria has been noted to come from both kidneys.[16, 17] This fact has not been satisfactorily explained. There is a tendency for the thrombosis to extend bilaterally if only 1 renal vein is involved initially.[16, 18]

Renal function in documented cases of renal-vein thrombosis may be normal initially[11] but may diminish rapidly.[5]

The clinical diagnosis of renal-vein thrombosis was initially suspected in this patient because of episodes suggestive of pulmonary embolization.

X-ray Findings

Although the disease can be suspected on clinical findings, X-ray examination is necessary for confirmation of the diagnosis. Scanlon[18] has listed the radiologic changes in renal-vein thrombosis:

1. Enlargement of the kidney outline, both parenchymatous and at the expense of the pelvis.
2. Distortion of the collecting system.
3. Irregularity of the proximal ureter and pelvis.
4. Thrombosis in the inferior vena cava.
5. Thromboembolism, such as pulmonary emboli.

Enlargement of the parenchymatous portion of the kidney shadow is due to edema. However, enlarged kidney size due to the same mechanism is a common finding in the nephrotic syndrome of other etiologies.[19] There is elongation of the infundibula and pressure defects on the calyces and pelvis of the kidney. This pseudocystic appearance of the calyces is also a result of edema of the kidney substance.[20]

The distortion and notching of the pelvis of the kidney and the upper one-third of the ureter has mimicked the appearance of ureteritis cystica and pyelitis cystica. It has sometimes been confused with pelvic and ureteral tumor. The notching was originally attributed to edema.[13, 20] It has been demonstrated, however, that it is due to collateral renal venous channels.[21, 22] Ureteral and pelvic notching are also seen in renal arterial occlusion,[21, 23] where it is due to the development of collateral arterial vessels.

X-ray studies of the inferior vena cava lead to a diagnosis of renal-vein thrombosis by inference. The failure to visualize "streamlining" in the inferior vena cava at the level of the renal veins at the time of vena cavography and the failure to visualize the renal veins with the use of the Valsalva maneuver do not prove renal-vein thrombosis.[24] Proof of patency of the renal veins may be obtained by successful selective renal-vein catheterization and venography. However, the failure to catheterize the renal vein, while highly suggestive, is not diagnostic of renal-vein thrombosis.[25] The hazard of knocking a portion of the thrombus loose is apparent but has not been reported in the literature.

Beres et al[26] have advocated needle puncture of the kidney and the blind localization by aspiration of a parenchymatous renal venous channel. Injection of contrast material into this venous channel allows definition of the large venous collaterals. The hazards of the procedure are obvious, and the technique has not found favor in the literature.

Injection of contrast material in the renal arteries with films programed to visualize the venous phase of the renal angiogram may yield definitive proof of renal-vein thrombosis.[22]

The X-ray and catheterization findings in this patient are consistent with the diagnosis of renal-vein thrombosis. The notching of the upper one-third of the ureter (figure 1) on the right side demonstrated by pyelography, and the demonstration of a thrombus in the inferior vena cava at the level of the renal veins by inferior vena cavography (figure 3) support the diagnosis of renal-vein thrombosis.

Pathology

The pathology of renal-vein thrombosis has been studied with both light and electronmicroscopy.[3, 27, 28]

Glomerular changes. The presence of diffuse basement-membrane thickening has been found universally. In one case studied by electronmicroscopy,[27] the basement membrane measured between 8,000 and 15,000 Angström units.

The thickening was due to the deposition of material between the lamina densa and the epithelial-cell foot processes. Vesicles were present in this deposited layer. Fusion of the foot processes was thought to be the result of proteinuria and not specific.

Although the appearance of the thickened basement membrane has occurred as early as the fourteenth day after the onset of renal-vein thrombosis,[14] the basement-membrane changes may occur after a longer interval. Brewer[29] illustrates a normal renal biopsy obtained from a patient $3\frac{1}{2}$ months after the onset of renal-vein thrombosis. Eight and one-half months following the first biopsy another biopsy showed thickening of the glomerular basement membrane.

Morris[17] found bilateral glomerular basement-membrane changes to be associated with unilateral renal-vein thrombosis. However, a recent article[30] noted only ipsilateral glomerular basement-membrane changes with unilateral renal-vein thrombosis.

Margination of the leukocytes in the glomerular capillaries have been described by Pirani[28, 31] in patients with renal-vein thrombosis. Resolution of some elements of the glomerular lesion has occurred in a 4-month period of treatment with anticoagulants.[28]

Tubular and interstitial changes. The tubular changes described by Panner[27] are nonspecific. Interstitial edema and later atrophy of the tubules and fibrosis of the interstitium with infiltration of chronic inflammatory cells have been described.[14, 28] Edema of the interstitium out of proportion to the degree of glomerular change is a histologic clue to the diagnosis of renal-vein thrombosis.[14, 30]

Renal veins. Recanalization in the major renal veins and the microscopic appearance of "webs" in the smaller renal venules have been noted[11] late after renal-vein thrombosis. The thrombosis may extend retrograde to the interlobular veins of the kidney in cases of primary renal-vein thrombosis or renal-vein thrombosis associated with inferior vena-caval thrombosis.[16] In patients with primary parenchymal renal disease the thrombosis may occur in the smaller venous radicles initially and extend toward the main renal veins.[4, 13]

The histologic findings of the kidney biopsy in this patient demonstrate basement-membrane thickening. The comment of the pathologist reviewing the sections is: "On a histologic basis it would be difficult, if not impossible, to distinguish these changes from the usual variety of mixed membranous glomerulonephritis. The glomerular changes appear as striking as the tubular changes. There is less interstitial edema than would be expected from a renal-vein thrombosis."

The changes reported in the literature and seen in this patient can only be considered confirmatory or at best suggestive of renal-vein thrombosis. Histologic changes on renal biopsy are not diagnostic of this disease entity.

Prognosis and Therapy

The prognosis depends on the underlying cause, the extent, and the speed of the thrombosis. Prognosis is directly related to the development of adequate collateral venous channels.

In a series collected from the literature, the average survival was 9 months after the onset of the nephrotic syndrome.[3] One-third of the deaths were due to uremia. Other major causes of death were congestive heart failure, pulmonary infarction, and coronary artery disease. In another compilation[10] 51 out of 65 patients were dead 2 years after the onset.

Prolonged survival has occurred. A survival of 8 years[17] has been documented in the literature. The longest survival reported is 25 years (quoted by Pollak[14] and reported by Shattuck). This patient had the nephrotic syndrome, renal-vein thrombosis, and inferior vena-caval thrombosis.

Nephrectomy was advocated and reported by Abeshouse.[13] The purpose was to prevent the spread of the thrombosis to the other kidney. He suggested, however, that the prime surgical attack should be upon the renal vein for the removal of the occluding thrombus and preservation of the kidney. At the time of his review (1945) such surgical therapy had not been attempted. Since then, when thrombectomy has been attempted,[31, 32] therapeutic failure has occurred despite good venous backflow observed at the time of surgery.

Steroids have been without effect[17, 30] in inducing a remission of the nephrotic syndrome, as have been steroids and nitrogen mustard combined.[33]

Anticoagulants were first suggested in 1944[34] and have been stressed as appropriate treatment more recently.[2] Complete remission of the nephrotic syndrome has been induced by anticoagulants.[10, 28] In 1 case treatment was started 8 years after the onset of the nephrotic syndrome.

Summary

A case of renal-vein thrombosis has been reported. The diagnosis was suspected clinically and confirmed by pyelography and angiographic studies of the inferior vena cava. A renal biopsy was nonspecific but was consistent with the diagnosis. The patient was treated with anticoagulants without effect for over 1 year. The literature has been reviewed.

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Chylothorax in the Newborn

Report of a Case Associated with Polyhydramnios

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A CASE OF CHYLOTHORAX in a newborn infant with a benign course with spontaneous recovery is presented here. The condition was associated with polyhydramnios in the mother. Chylothorax in the newborn is an uncommon condition which may produce respiratory distress, cyanosis and subcostal and intercostal retractions. The breath sounds are diminished and there is dullness on percussion on the side involved. These findings are noticed at or shortly after birth, although at times they may not appear until weeks or months later.

Chylothorax has been known since the 17th century,[1] but it was not until 1926 when Stewart and Linner[2] reported the first case in the newborn. Since then few cases have been reported and, as far as we can determine, less than 34 cases have appeared in the English literature and not many more in the world literature.

Report of Case

A full-term female Negro newborn infant was delivered to a gravida IX, Para 8 mother after a normal spontaneous delivery. The mother was in good health. She weighed 200 lbs. Pregnancy was uneventful, but polyhydramnios was diagnosed at delivery.

The baby weighed 8 lbs., 11 ozs. The condition at birth was described as good, and Apgar score at 1 minute was 9. She was 21.5 inches in length, her head circumference was 35 cm., and chest circumference, 33 cm. The heart rate was 122 per minute with no audible murmurs. The head was normocephalic; the eyes, ears and the oral cavity were normal. The respiratory rate was 60 per minute with diminished breath sounds on the right side. The abdomen was soft with no hepatosplenomegaly. The extremities were normal. Sucking, Moro and grasp reflexes were normal.

The baby was found to have a dusky color at 1 hour. There was moderate respiratory distress with subcostal and intercostal retraction. Chest roentgenogram revealed bilateral homogeneous density. The infant was placed in an incubator with oxygen and high humidity and given intravenous fluids when 24 hours of age.

The hematocrit was 59 per cent. Urinalysis was normal. The serum electrolytes were: Na, 148; K, 6.1; Cl, 115 and CO_2, 19 mEq./L; the fasting blood sugar was 80 mg./100 ml.; total serum protein was 6.21 Gm./100 ml., with 4.25 Gm./100 ml. as albumin and 1.96 Gm./100 ml. as globulin.

The baby's condition improved gradually and another chest roentgenogram at 4 days of age showed diminution of the homogeneous density with evidence of fluid on the right side. The baby was followed closely, and serial chest roentgenograms showed gradual diminution of the fluid with increased aeration. Thoracentesis was performed at 11 days of age just before her scheduled feeding. It revealed 35 ml. of a yellowish white fluid from the right side. Protein electrophoresis and lipid determination were performed on the serum and on the fluid obtained (table 1 and figure 1). The infant continued to improve, and at 20

TABLE 1: Lipid Content and Protein Electrophoresis of the Serum and Chylous Fluid—Fasting and Postprandial Values

SOURCE	Total Lipids (mg. %)	Cholesterol (mg. %)	Triglycerides (mg. %)	Phospholipids (mg. %)	Total Proteins (Gm. %)	Albumin (Gm. %)	Alpha I Globulin (Gm. %)	Alpha II Globulin (Gm. %)	Beta Globulin (Gm. %)	Gamma Globulin (Gm. %)
Serum—fasting	452	124	72	200	6.2	2.38	0.47	0.61	1.07	1.6
Serum—postprandial	554	167	110	202	6.18	2.67	0.28	0.76	0.81	1.6
Chyle—fasting	1,373	64	123	1,157	4.25	1.62	0.26	0.43	0.82	1.1
Chyle—postprandial	2,888	92	235	2,520	4.35	1.80	0.35	0.46	0.70	1.0

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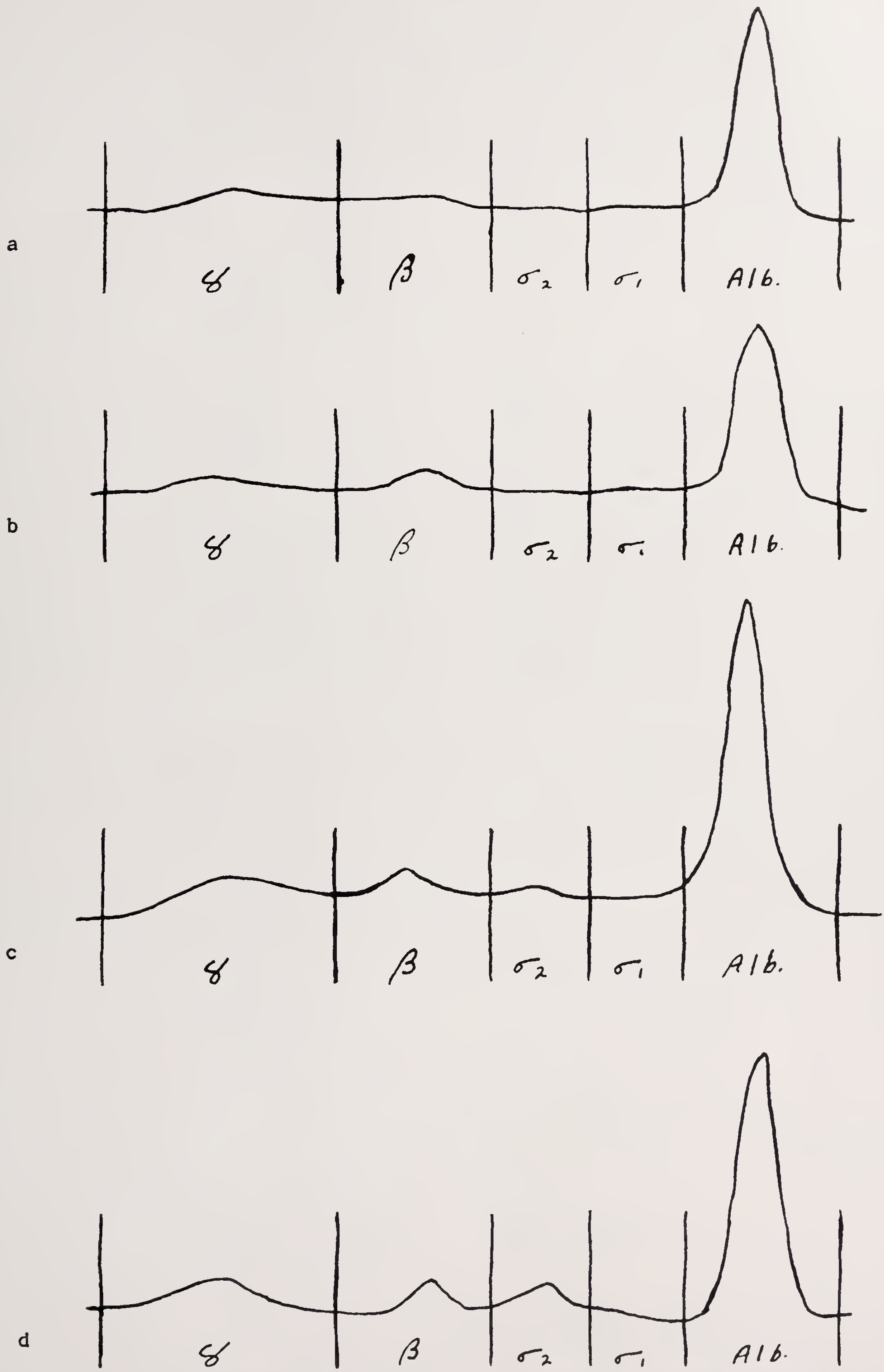

FIG. 1. Protein electrophoresis: chyle—a, fasting and b, postprandial; serum—c, fasting and d, postprandial.

days of age a second thoracentesis was performed, this time 1 hour after feeding. Thirty ml. of a creamy white fluid was obtained from the same side. It had a specific gravity of 1.027 and contained large numbers of chylomicrons. Sudan stain for fat oval bodies was positive. The lymphocyte count of the fluid was 60,000 per cu. mm. Protein electrophoresis was performed and lipid contents of the serum and the chylous fluid determined. The findings are shown in table 1 and figure 1. A chest roentgenogram at 21 days of age showed no evidence of reaccumulation of the fluid in the chest. The infant was discharged at 23 days of age in good condition.

Discussion

Chylothorax is known to occur at any age, but its presence in the neonatal period is quite rare. It is usually associated with tumors and trauma. Chyle is a lymphatic fluid that contains a large amount of emulsified fat, derived from food. Its color and homogeneity depend on its fat content which varies with the amount of fat ingested. The age of the child and the time and type of feeding may influence the character of the chylous fluid. In the fasting state and in the first day or 2 of life the lymph coming from the omentum is a clear transparent fluid which may assume the milky or white opalescent character of chyle after the introduction of milk feeding.

In the case presented here 2 thoracenteses were done quite some time after the introduction of evaporated milk formula. The first tap was done just before a scheduled feeding and the second 1 hour after a feeding. The fluid obtained in the first tap had a lighter color, was less opalescent, and contained about half the amount of total lipids found in that obtained in the second thoracentesis. The protein content of the fasting and postprandial fluids did not differ significantly, but in both it was less than that of the serum.

Table 2 gives a summary of the cases of neonatal pleural effusion in the English literature.[2–25] Cases 1 and 8 are included, since the diagnosis was made shortly after the neonatal period, and it is felt that the condition was present early in that period but manifested later.[3, 9] In 24 cases the fluid was identified as chyle. In the remaining 5 cases the fluid was described as orange, clear yellow, or straw-colored.[10, 20, 21, 22, 24] In 4 of these 5 cases thoracentesis was performed early in the first few days of life, and it is thought that the fluid could have assumed the character of chyle if thoracentesis were done at a later date.[20, 21, 22, 24] This view was expressed by Schaffer,[24] who stated in his case that the fluid could have changed into chyle if thoracentesis was repeated later, after the establishment of milk feeding. Perry et al[21] thought that the classification of cases of neonatal pleural effusion into chylous and nonchylous is unjustifiable and that the nature of the fluid depends on the age and time of diagnosis. This point is illustrated further in the case presented here, which shows variation in the character of the fluid obtained before and after feeding.

The etiology of neonatal chylothorax is not quite clear. Avery[26] stated that "this unusual condition in the newborn is as puzzling as it is rare." In the few cases that resulted in death and an autopsy was performed the findings were inconclusive and variable. In 4 out of 6 cases death resulted. No thoracic duct was identified at autopsy.[2, 5, 6, 8] In 2 cases there was leakage of fluid in the posterior mediastinum just above the diaphragm.[2, 6] Bornhurst and Carsky[22] injected methylene blue into the beginning of the thoracic duct and found no leakage into the pleural space. McKendry and Lindsay[15] found multiple dilated lymphatic channels with failure of the peripheral channels to communicate with the larger lymphatics causing back pressure and thus leakage of lymph, but they emphasized that no actual break in the thoracic duct was responsible for the chylothorax. Feinerman et al[17] found in an autopsy performed in a 4 months' old infant atresia of the thoracic duct and dilatation of the cisterna chyli. This child had, in addition to chylothorax, chylous ascites which was apparent at birth. Kirkland[27] reported a child with lymphedema of the right leg noticed at birth and at the age of 4 years found to have bilateral chylothorax. At autopsy multiple fistulae along the lower half of the mediastinal pleura were found which communicated with large dilated retroperitoneal lymph channels. Kelly[19] found aplasia and congenital atresia of the thoracic duct, lymphangiomatous lesions producing block of the central lymphatics, and congenital lymphatic malformations in few children with chylothorax with ages ranging from 6 weeks to 9 years.

Other sources of information regarding the pathogenesis of neonatal chylothorax are based on the findings seen after thoracotomy. In one of the cases reported by McKendry and Lindsay[15] generalized serous weeping from the entire pleural surface was noticed, but the source of chyle could not be identified. Randolph and Gross[16] thought that 2 types of congenital anomalies of the thoracic duct may be encountered in neonatal chylothorax. The first anomaly consists of a series of fistulae between the thoracic duct and the pleural space, allowing free access of chyle into the pleural cavity and causing respiratory distress immediately after

TABLE 2: Cases of Pleural Effusion in the Newborn

CASE	Age	Sex	Signs	Side	Number of Aspirations	Nature and Amount Aspirated	Outcome	Autopsy If Performed
1. Pisek[3] 1917	2 months	F	Rapid, difficulty in breathing	Right	2	? Chyle	Recovered	
2. Stewart and Linner[2] 1926	4 days	M	Respiratory distress, cyanosis	Right	14	1,155 c.c. chyle	Died	Pressure on abdominal viscera resulting in escape of chylous fluid from a point in the posterior mediastinum just above diaphragm but thoracic duct not identified.
3. Janet et al[4] 1936	21 days	M	Respiratory distress	Right	2	2,500 c.c. chyle	Recovered	
4. Hilgenberg[5] 1939	4 days	F	Difficult breathing	Right	None		Died	Chyle found in thoracic cavity;thoracic duct not identified.
5. Everhart and Jacobs[6] 1939	15 days	M	Extreme cyanosis	Right	Done every 24 to 36 hours for 21 days	? Chyle	Died	Leakage of chyle from the posterior mediastinum 1½ inches above the diaphragm but thoracic duct not identified.
6. Wessel[7] 1944	2 weeks	M	Difficult breathing	Right	13	1,278 c.c. chyle	Recovered	
7. Forbes[8] 1944	2 weeks	F	Dyspnea; cyanosis	Bilateral	42	5,300 c.c. chyle	Died	No thoracic duct or cisterna chyli identified.
8. Watson and Foster[9] 1944	7 weeks	F	Irregular breathing; diarrhea	Right	14	2,245 c.c. chyle	Recovered	
9. Cugell and Scherl[10] 1949	15 hours	M	Subcostal retraction	Right	5	? Turbid, orange to dark orange	Recovered	
10. Sakula[11] 1950	2 days	M	Rapid respiration	Right	2	130 c.c. chyle	Recovered	
11. Martiniz[12] 1950	4 days	M	Difficult breathing	Left	?	?	Recovered	
12. Blood and Fairchild[13] 1953	3½ weeks	F	Difficult breathing	Right	5	590 c.c. chyle	Recovered	
13. Stirlacci[14] 1955	1 hour	F	Respiratory distress; diarrhea	Right	3	? Chyle	Recovered	
14. McKendry and Lindsay[15] 1957	Birth	F	Dyspnea; diarrhea	Left	7	565 c.c. chyle	Recovered	
15. McKendry and Lindsay[15] 1957	Birth	M	Panting, shallow respiration	Left	3	350 ml. chyle	Recovered. Thoracotomy showed serous weeping of entire pleural surface; source of chyle not identified.	
16. McKendry and Lindsay[15] 1957	Birth	F	Edema of leg				Died: had chylous ascites and chylothorax	Multiple dilated lymph channels with failure of peripheral channels to communicate with larger lymphatics, causing back pressure and leakage of lymph. No actual break in thoracic duct.
17. Randolph and Gross[16] 1957	1 hour	M	Difficult breathing	Left	10 + thoracotomy	495 ml. chyle	Recovered	
18. Randolph and Gross[16] 1957	Birth	F	Tachypnea; cyanosis	Left	6 + thoracotomy	? Chyle	Recovered	
19. Feinerman et al[17] 1957	5 days	?	Difficult breathing	Left	1	165 ml. chyle	Recovered	
20. Boles and Izant[18] 1960	4 weeks	M	Grunting respiration; cyanosis	Right	27	1,995 ml. chyle	Recovered	
21. Boles and Izant[18] 1960	Birth	M	Cyanosis; shallow breathing	Right	5	316 ml. chyle	Recovered	
22. Kelly[19] 1960	Birth	M	Dyspnea; large abdomen	Right			Recovered	
23. Stephan and Otto[20] 1961	Birth	M	Difficulty in establishing breathing	Bilateral	1	75 ml., clear yellow	Recovered	
24. Perry et al[21] 1963	58 hours	M	Respiratory distress	Right	2	350 ml., turbid orange	Recovered	
25. Bornhurst and Carsky[22] 1964	Birth	F	Dyspnea	Bilateral	3	85 ml., clear yellow	Died suddenly at 15 hours of age	Methylene blue injected into origin of thoracic duct showed no leakage into pleural space.
26. Bornhurst and Carsky[22] 1964	Shortly after birth	M	Respiratory distress; cyanosis	Left	1	160 ml.; 30 ml. chyle	Recovered	
27. Decancq[23] 1965	1 day	?	Respiratory distress	?	1	10 ml. chyle	Recovered	
28. Schaffer[24] 1965	Birth	M	Respiratory distress	Right	1	80 ml., straw-colored	Recovered	
29. Gwinn[25] 1968	5 days	M	Respiratory distress	Right	?	?	?	

birth. The second type of malformation is confined to the wall of the thoracic duct where there may be 1 or multiple openings or areas of weakness which are unable to stand the strain imposed on the fetus during the process of birth. In this case the chyle will be confined in the mediastinum initially, during which time the newborn infant manifests no difficulty until chyle breaks out into the pleural space, producing respiratory symptoms.

That fistulae may be the underlying cause was also proposed by Nix et al,[28] who suggested that chylothorax is a complication of internal lymph fistulae. Bornhurst and Carsky[22] believed that immaturity of the lymphatic system is the cause of accumulation of chyle in the body cavities. Others such as Pisek[3] and Stirlacci[14] thought that trauma associated with birth is the cause of neonatal chylothorax.

From the above review it seems more likely that the syndrome of neonatal chylothorax is the result of multiple etiologic factors. Among these, congenital malformation of the thoracic duct, such as atresia or absence, fitulae between the thoracic duct and pleural surface, malformations of the other lymphatic channels, and trauma are the most important ones. In most cases, however, no specific abnormality could be encountered, and recovery is complete with conservative therapy. To these cases the name spontaneous chylothorax has been applied. It is possible that immaturity of the lymphatic system as proposed by Bornhurst and Carsky is the underlying cause for the so-called spontaneous chylothorax. These constitute the majority of cases of chylothorax in the newborn.

In the case presented here polyhydramnios was a concomitant finding. Polyhydramnios is known to be associated with various congenital anomalies of the newborn, including anencephaly and esophageal and duodenal atresia. Whether this bears any relation to chylothorax or is an accidental finding was not determined.

The diagnosis of this rare condition is important. It could be dangerous and fatal if the diagnosis is missed and thoracentesis not performed at the right time. Chylothorax should be included in the differential diagnosis of respiratory distress in the newborn. The roentgenologic picture is quite characteristic, although at times it may be confused with primary atelectasis from which it should be differentiated promptly.[22] In primary atelectasis both sides are involved, and there is narrowing of the intercostal spaces with slanting of the ribs downward from behind to front and the diaphragms are elevated. Chylothorax is usually limited to one side, and here the intercostal space is wider and the ribs horizontal. The diaphragms may be depressed.

The management is conservative in most cases. Thoracentesis should be done whenever there is respiratory distress to relieve the symptoms and to remove the accumulating chyle. If the amount of fluid removed is large and repeated taps are necessary over a long period of time, attention should be paid to provide enough protein and calories. Supplemental amounts of fat-soluble vitamins must be given. If accumulation of chyle persists in spite of repeated thoracenteses, surgical approach may be necessary, and thoracotomy may reveal an underlying abnormality which may be amenable to surgical correction. It must be reëmphasized that great care must be taken to avoid injury to the lung or introduction of air during thoracentesis because tension pneumothorax may develop with serious outcome. Decancq[23] suggested the use of a thoracic tube with continuous suction and negative pressure. Roy et al[29] in their review of chylothorax state that aspiration is adequate in cases caused by congenital anomalies. Simple thoracentesis is all that is needed in the majority of cases.

Summary

A case of neonatal chylothroax with spontaneous recovery has been reported. The condition was associated with polyhydramnios. All of the cases of chylothorax and pleural effusion in the newborn which have been reported in the English literature have been reviewed and the possible etiologic factors discussed. In the majority of cases no cause was determined and the name spontaneous chylothorax was applied. These cases may be due to immaturity of the lymphatic system, allowing leakage of the lymph into the pleural cavity. Congenital anomalies of the thoracic duct and other lymphatic vessels as well as trauma may be the underlying cause in a small percentage of cases. Treatment has been discussed, and it appears that conservative therapy with thoracentesis as necessary is the method of choice in the majority of cases.

We are indebted to Mr. Jack Thomas who performed the biochemical studies.

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Extruded Upper Thoracic Disc Causing Horner's Syndrome

Report of a Case

LT. COLONEL WILLIAM M. HAMMON, MC, USA*

THE LATERAL PROTRUSION of an intervertebral disc in the thoracic region is uncommon. Love and Schorn[1] reported 61 cases of thoracic disc protrusion in 1965 of which only 14 were laterally placed. Herniation usually occurs in the lower thoracic area, most frequently causing spinal-cord compression for which surgical intervention is often ineffective in reversing cord damage and has also been recognized to have caused increased neurologic deficit.

Rupture of upper thoracic discs is considered rare.[2, 3] Those occurring laterally not causing cord compression or damage but causing a syndrome of radicular impairment are even more unusual. Add to this manifestations of upper sympathetic nervous-system involvement and an even more noteworthy situation exists.

Report of Case

The patient was a 33-year-old white man who noted the spontaneous onset of left scapular area pain 2 months prior to admission to Walter Reed General Hospital. This occurred during mild exercise with his young child who was climbing on his back. The pain was not severe until 2 weeks prior to admission when it radiated down the inner aspect of the left arm to the wrist and was associated with marked impairment in sensation to touch and pain on the volar aspect of the fourth and fifth fingers and inner aspect of the hand, forearm, and upper arm. Outpatient traction, heat, and local scapular corticosteroid injections were without benefit.

On admission in February 1967 the patient's positive physical findings included a definite narrowing of the left palpebral fissure, a left pupil diameter of 3 mm. with a 5 mm. pupil on the right, anesthesia to pin prick and touch over the ulnar aspect of the left forearm, hypalgesia and hypesthesia over the ulnar side of the left hand and fifth digit and the ulnar side of the fourth digit, and tenderness over the midscapular-paravertebral area on the left. Neck flexion caused acute dysesthesias and pain into the above-noted areas of sensory impairment. There were no abnormal reflexes in any extremity, no signs of spinal-cord compression, and no gross demonstrable motor loss.

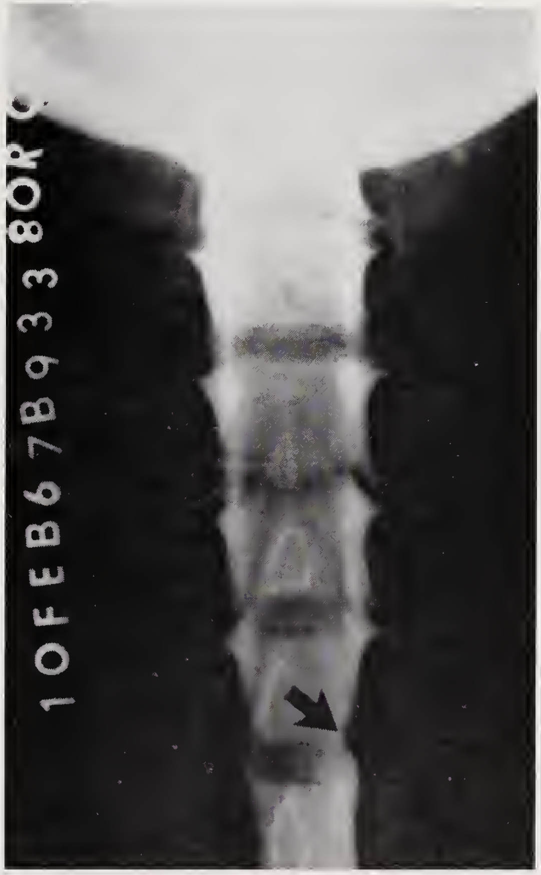

FIG. 1. Cervicothoracic myelogram with arrow showing left T_{1-2} defect.

Laboratory data included normal peripheral blood counts and chemistries, urinalysis, and X-ray films of the cervical and thoracic vertebrae and chest. The cerebrospinal fluid withdrawn prior to instillation of the positive contrast myelographic medium contained 3 lymphocytes per cu. mm., 82 mg. of total protein, and no bacterial growth on culture. The myelographic study (figure 1) revealed a persistent epidural defect at the T_{1-2} level on the left.

A hemilaminectomy of T_1 was performed on the left, and a small free fragment of degenerative fibro-

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Erratum: In the paper entitled, "Clinical Studies in the Use of Oral Diazoxide in Postsurgical and Functional Hypoglycemia" by John K. Wales, M.D., Heinrich Spechtmeyer, M.D., Josef K. Viktora, Ph.D., and Frederick W. Wolff, M.D., which appeared in the September 1968 issue of the Medical Annals (Volume 37, No. 9, pages 460–468), the authors regret there was a transposition of symbols in the legends for figures 1–5.

cartilage was easily removed laterally from under the T_1 nerve root. No further excision of disc material from the intervertebral space was attempted.

Postoperatively the patient's pain abated completely, but his profound dermatomal sensory loss persisted as well as the Horner's syndrome. He was returned to full duty, and evaluation 1 year later revealed a mild persistence of the left ptosis and miosis and only a small area of hypalgesia and hypesthesia of the left forearm.

Comment

Review of the literature reveals only 7 cases of rupture of a thoracic disc at T_{1-2} and only the case of Horwitz et al[4] in 1955 had an associated Horner's syndrome. Their case was also readily diagnosed by myelography, and curative surgery was performed.

Besides noting the presence of an upper thoracic radicular syndrome and the relief of symptoms by appropriate surgery, it is perhaps of additional importance to again note that the presence of intracanalicular masses in the thoracic area is even more critical than those in the cervical and lumbar areas because of a narrower canal and marginal collateral vascular supply. When a syndrome suggesting thoracic root or cord compression is seen, early investigation is warranted to prevent the development of otherwise irreversible spinal-cord damage.

Summary

A second case of upper thoracic disc protrusion, manifested by radiculitis, radiculopathy, and a Horner's syndrome, has been reported.

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Continuing Medical Education—
Chiazze and Mattingly

(*Continued from page 522*)

Given that we have identified some mechanisms for carrying on continuing education, we must be aware of some of the difficulties involved and what must be done to insure success. Table 7 indicates a distribution of factors affecting participation in continuing education. Nearly 60 per cent of the physicians responding indicated that there was some difficulty in participating in some way. As might be expected, the most frequent reason given for not participating was that such participation would interfere with time committed to patients (which also is a part of the combination of factors). This indicates rather clearly that programs, in order to be utilized, must be available at times which do not conflict with the care of patients, e.g., evenings or weekends. Such times, of course, are a burden for those who assume the responsibility for providing continuing education and training, but such burdens must necessarily be assumed if the programs are to be successful. We have some indication of the kinds of continuing education and programs that will be successful and some indication of why such programs have not been successful. If we are serious about what we wish to accomplish through continuing education, it is clear that every effort must be made to provide such programs at the convenience of those to whom the continuing education is directed, rather than the convenience of those who are providing the continuing education. Furthermore, it is necessary to build in some mechanism to evaluate the benefit to patients. From the viewpoint of the Regional Medical Program the success of a program of continuing education must be measured in terms of what it does for the patient, not what it does for the physician.

We wish to thank the physicians who responded to our questionnaire and made it possible to provide the data in this report.

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Warnings—Watch for the earliest manifestations of thrombotic disorders (thrombophlebitis, cerebrovascular disorders, pulmonary embolism, retinal thrombosis); if present or suspected discontinue the drug immediately.

British studies reported in April 1968[1,2] estimate there is a seven- to tenfold increase in mortality and morbidity due to thromboembolic diseases in women taking oral contraceptives. In these controlled retrospective studies, involving 36 reported deaths and 58 hospitalizations due to "idiopathic" thromboembolism, statistical evaluation indicated that the differences observed between users and non-users were highly significant. The conclusions reached in the studies are summarized in the table below:

Comparison of Mortality and Hospitalization Rates Due to Thromboembolic Disease in Users and Non-Users of Oral Contraceptives in Britain.

Category	Mortality Rates		Hospitalization Rates (Morbidity)
	Age 20-34	Age 35-44	Age 20-44
Users of Oral Contraceptives	1.5/100,000	3.9/100,000	47/100,000
Non-Users	0.2/100,000	0.5/100,000	5/100,000

No comparable studies are yet available in the United States. The British data, especially as they indicate the magnitude of the increased risk to the individual patient, cannot be applied directly to women in other countries in which the incidences of spontaneously occurring thromboembolic disease may differ.

Discontinue medication pending examination if there is sudden partial or complete loss of vision, or sudden onset of proptosis, diplopia or migraine. Withdraw medication if papilledema or retinal vascular lesions are found.

Since the safety of Ovulen in pregnancy has not been demonstrated, it is recommended that pregnancy be ruled out for any patient who has missed two consecutive periods before continuing the contraceptive regimen. If the patient has not adhered to the prescribed schedule the possibility of pregnancy should be considered at the first missed period.

A small fraction of the hormone agents in oral contraceptives has been identified in the milk of mothers receiving these drugs. The long-range effect to the nursing infant cannot be determined at this time.

Precautions—Pretreatment physical examination should include special reference to the breasts and pelvic organs, and a Papanicolaou smear.

Endocrine and possibly liver function tests may be affected by Ovulen. Therefore, it is recommended that such tests if abnormal be repeated after the drug has been withdrawn for two months.

Pre-existing uterine fibromyomas may increase in size under the influence of progestogen-estrogen preparations.

Because these agents may cause some degree of fluid retention, conditions which might be influenced by this factor, such as epilepsy, migraine, asthma, cardiac or renal dysfunction, require careful observation.

In breakthrough bleeding, and all irregular vaginal bleeding, consider nonfunctional causes. Adequate diagnostic measures are indicated in undiagnosed vaginal bleeding.

Carefully observe patients with a history of psychic depression and discontinue the drug if severe depression recurs.

Any possible influence of prolonged Ovulen therapy on pituitary, ovarian, adrenal, hepatic or uterine function awaits further study.

A decrease in glucose tolerance has occurred in a significant percentage of patients on oral contraceptives. The mechanism of this decrease is obscure. For this reason, diabetic patients should be observed carefully while receiving Ovulen.

Because of the effects of estrogens on epiphyseal closure Ovulen should be used judiciously in young patients in whom bone growth is not complete.

The age of the patient constitutes no absolute limiting factor, although Ovulen therapy may mask the onset of the climacteric.

The pathologist should be informed of Ovulen therapy when relevant specimens are submitted.

Adverse Reactions—A statistically significant association has been shown between use of oral contraceptives and the following serious adverse reactions: thrombophlebitis, pulmonary embolism.

Although available evidence is suggestive of an association, such a relationship has been neither confirmed nor refuted for the following serious adverse reactions: cerebrovascular accidents, neuro-ocular lesions, e.g., retinal thrombosis and optic neuritis.

The following adverse reactions are known to occur in patients receiving oral contraceptives: nausea, vomiting, gastrointestinal symptoms (such as abdominal cramps and bloating), breakthrough bleeding, spotting, change in menstrual flow, amenorrhea during and after treatment, edema, chloasma or melasma, breast changes (tenderness, enlargement, secretion), change in weight, changes in cervical erosion and cervical secretions, suppression of lactation when given immediately post partum, cholestatic jaundice, migraine, allergic rash, rise in blood pressure in susceptible individuals, mental depression.

Although the following adverse reactions have been reported in users of oral contraceptives, an association has been neither confirmed nor refuted: anovulation post treatment, premenstrual-like syndrome, changes in libido, changes in appetite, cystitis-like syndrome, headache, nervousness, dizziness, fatigue, backache, hirsutism, loss of scalp hair, erythema multiforme and nodosum, hemorrhagic eruption, itching.

The following laboratory results may be altered by oral contraceptives: hepatic function: increased sulfobromophthalein and other tests; coagulation tests: increase in prothrombin, Factors VII, VIII, IX and X; thyroid function: increase in PBI and butanol extractable protein bound iodine, and decrease in T^3 values; metyrapone test; pregnanediol determination.

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President's Page

THE SCIENTIFIC ASSEMBLY

On November 21, 22 and 23 the Medical Society of the District of Columbia will present what we believe will prove to be the finest Scientific Assembly in its long and distinguished history. Moreover, those who have studied the program agree that it will be one of the best meetings of general medical and surgical interest ever offered in the City of Washington. At considerable effort only speakers and panelists of unquestioned authority in their respective fields have been selected from every part of the country.

Subjects to be discussed include peptic ulcer, hiatal hernia, infectious diseases, carcinoma of the breast, drug abuse, and current concepts of resuscitation. Among the discussants will be Drs. Oliver Cope, Franz J. Ingelfinger, T. Wilkins Davis, Robert M. Zollinger, Harry F. Dowling, Edward F. Lewison, and many others. Perhaps the highlight of the Assembly will be presentations and a panel discussion on organ transplantation of the heart, liver, and kidney by Drs. Shumway, Starzl, Harrison, and Schwartz. This will end in an evening panel, open to the public, on the moral, religious, and legal aspects of organ transplantation, moderated by Mr. William Hines, Washington Editor of the World Book Science Service. Panelists will include Drs. Shumway and Starzl, the Reverend Dr. Joseph Fletcher, and Dr. Paul D. Cantor, Professor of Medical Jurisprudence at Georgetown University Law Center.

It is in many ways unfortunate that our Medical Society holds only one large scientific meeting each year. Specialty groups and hospital staff meetings have replaced the Society forums of the past. It has been suggested that perhaps our one annual scientific session should be abolished. To prove that this is neither necessary nor advisable, we have secured this distinguished group of talented teachers for 1968. The time is well chosen; the distance, short; and the attraction, great. Don't miss it!

William S. McCune

Editorials

An Outstanding Program for the 1968 Scientific Assembly

The Committee on Continuing Education has as one of its major responsibilities the preparation of the program for the Medical Society's Annual Scientific Assembly. This Committee has worked hard and long, and its members believe that this year's program is indeed outstanding.

The posture of postgraduate medical education in this community has significantly changed in the past several decades. No longer does the Medical Society play a prime role in the continuing education of Washington physicians. Many, if not most, of our community hospitals have appointed directors of medical education who are responsible for ongoing weekly or monthly educational programs. Many of these institutions have, in essence, undertaken the primary role in the continuing education of their attending physicians. What role the Medical Society should play in the future remains to be seen. However, it was the decision of this year's Committee that an outstanding group of experts be invited to speak on a broad range of subjects of medical interest. We hope that you will enjoy this year's sessions.

Howard E. Ticktin, M.D.
Chairman, Committee on Continuing Education

Wanted: Physicians Interested in Auto Accident Prevention

Teams of physicians and police traffic officers have been proposed as a means of collecting vitally needed scientific data from auto accidents.

Dr. H. A. Fenner, Jr., of Hobbs, New Mexico, Chairman of the American Medical Association's Committee on the Medical Aspects of Automotive Safety, points out that every traffic accident presents an opportunity to discover clues to prevention.

"Most accident reports at the present," Dr. Fenner said, "are related to law enforcement rather than to research-oriented data collection.

"If we had a corps of knowledgeable, trained, and qualified physicians and police officers to provide accurate, scientific reports on a large number of accidents, automotive engineers would have the necessary data to translate into safer motor vehicles and highways."

Many of the safety devices on present-day automobiles and highways have resulted from traffic accident research.

The AMA Committee, along with the American Association for Automotive Medicine, is studying the possibility of working with one or more universities in the establishment of intensive courses in accident investigation. Preliminary discussions have indicated that such a course would be of 3 days' duration and would be limited to 200 teams, each consisting of a physician and a member of a police traffic department.

The important need at the moment is to determine if enough physicians are interested in participating in such an effort. Physicians who are interested should send their name, address, age, medical college, and field of practice to the AMA Committee on Medical Aspects of Automotive Safety, 535 North Dearborn Street, Chicago, Ill. 60610.

This proposal has real merit. I believe that much can be learned from such data, and it is my hope that Washington physicians will want to participate in this program.

Herbert S. Gates, M.D.
Chairman, Traffic Committee

ANNUAL BUSINESS MEETING

Wednesday, November 13, 1968, 5:30 p.m.—Medical Society Building
Refreshments to follow business session.

Community Health Week 1968

The theme for Community Health Week, to open October 20, 1968, is "Good Health and Physical Fitness Go Hand in Hand." Under the direction of Drs. Nelson G. Goodman and Donald D. Haut a Health Exposition will be conducted in the Medical Society's building. Multiple studies—blood typing, chest X-rays, audiometry, blood pressure screening, EKG, blood sugar, and serology—will be done on all comers. The ophthalmologists are being contacted by Dr. Mervin H. Zimmerman to do glaucoma testing with the assistance of the Prevention of Blindness Society.

The Speakers Bureau, under Dr. Myer H. Stolar, will provide speakers on health topics for community organizations plus TV and radio spot appearances.

It is anticipated too that the Medical Society will demonstrate the way to achieve better physical health through physical fitness. Don't be surprised, therefore, if you see Senator Proxmire, of Wisconsin, leading Medical Society members on a jog down Pennsylvania Avenue. All those interested in joining The Jog (from Washington Monument to the Medical Society Building) on Sunday morning, October 20, at 10 a.m., telephone the Society's Executive Offices, 223-2230.

Warren D. Brill, M.D.
Chairman, Committee on Public Information and Education

Antihemophilic Factor Concentrate from Red Cross Plasma

An agreement for the production of a new and costly blood fraction from Red Cross plasma has been reached between the American Red Cross and Hyland Laboratories Division of Baxter Laboratories, Inc. It will make available Antihemophilic Factor Concentrate for the treatment of certain critical cases among the Nation's estimated 30,000 hemophilia victims.

Those afflicted with this condition have a tendency to bleed profusely from even the slightest wound. AHF is considered the most effective means of arresting bleeding, but supplies are still limited and production so expensive that its application usually is reserved for severe hemorrhage or use during pre- and postoperative care. In fact, until the advent of AHF most surgical procedures could not be attempted.

Other effective agents are available for less serious bleeding. They include fresh frozen single-donor plasma, cryoprecipitates, and some commercially prepared products.

Under the agreement between the Red Cross and Hyland, the AHF will be produced for Red Cross under the process presently licensed by the National Institutes of Health. It will be packaged in a 600-unit AHF vial suitable for intravenous administration.

The Red Cross is encouraging all of its 59 Blood Centers across the country to increase their participation in the AHF plasma program. The initial supply will be allocated to those Centers which have been participating in the Red Cross research program. Red Cross research efforts will continue to examine methods for improving production yields, product quality, clinical uses, etc., of Antihemophilic Factor Concentrates.

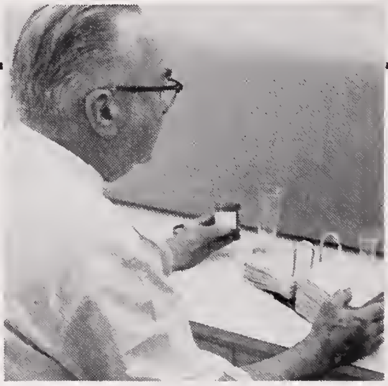
The American Red Cross is the largest single blood collection agency in the world. It provides an average of 3,000,000 units each year for use by civilian and military hospitals and medical research laboratories.

Because of the limited Red Cross supply of AHF and because the product is unfamiliar to most physicians, medical consultants will be appointed for each participating Blood Center to evaluate applications for use of the fraction.

As it does with other blood derivatives, the Red Cross will make AHF Concentrate available to patients for partial reimbursement of the cost of producing the plasma and processing and shipping the fraction.

The processing requires sophisticated and expensive equipment and procedures. An indication of the stringent controls necessary in AHF production is the procedure for donating and immediate processing. The Red Cross said the blood must be drawn from the donor within an 8-minute span. Within the next 4 hours the plasma must be separated from the red blood cells and then frozen. It must then be packaged and shipped in a frozen state to the processor where the actual fractionation process takes place.

Count the Prescriptions filled by Peoples Drug Stores and you get 100 million times people have counted on Peoples



We have passed the hundred million mark. That's the number of prescriptions filled by all Peoples Drug Stores since we opened our doors in 1905. We say this with great pride, since this impressive figure illustrates an impressive public confidence. Because behind all prescribed medicine at Peoples is confidence — in the physician who prescribes, the manufacturer who supplies, and the pharmacist who fills the prescription. At Peoples, nothing is more important to us than this confidence.

PEOPLES
PRESCRIPTION DRUG STORES

OFFICIAL PROGRAM

1968 Scientific Assembly
The Medical Society of the District of Columbia
WASHINGTON HILTON HOTEL

Scientific Program

THURSDAY MORNING, NOVEMBER 21, 1968
(All Meetings in the International Ballroom West)

Howard E. Ticktin, M.D., *Presiding*
General Chairman, 1968 Scientific Assembly

9:00 Welcome by **William S. McCune, M.D.,** President, The Medical Society of the District of Columbia

9:15 **PANEL ON BREAST CARCINOMA**

Moderator:

William Newman, M.D.
Professor of Pathology, George Washington University School of Medicine

Panelists:

Oliver Cope, M.D.
Professor of Surgery, Harvard Medical School; Visiting Surgeon, Massachusetts General Hospital, Boston

Ruth J. Guttmann, M.D.
Professor of Radiology, Columbia University College of Physicians and Surgeons

Edward F. Lewison, M.D.
Chief, Breast Clinic, Johns Hopkins Hospital, Baltimore

Mortimer Lipsett, M.D.
Chief, Endocrinology Branch, National Cancer Institute

10:25 Recess to Visit Exhibits

10:45 **SELECTED CASES IN INFECTIOUS DISEASES**

Moderator:

Harry F. Dowling, M.D.
Professor and Head, Department of Medicine, University of Illinois College of Medicine

Case 1: Pyelonephritis
Case 2: Bacterial Endocarditis
(Case presentations will be made by the chief resident physicians from 2 area hospitals.)

11:50 Recess for Lunch—Visit Exhibits

PROGRAM APPROVED BY AAGP

The Assembly's Scientific Program has been approved by the American Academy of General Practice for its members for a maximum of 15 hours' credit under Category I.

ACKNOWLEDGMENT: The Committee on Arrangements expresses its appreciation to Mutual of Omaha Insurance Company, Joseph E. Jones, Administrator, for contributing to the printing of the Official Program.

THURSDAY AFTERNOON, NOVEMBER 21

Frank S. Bacon, M.D., *Presiding*
President-elect, The Medical Society of the District of Columbia

2:00 **GASTROINTESTINAL SERIES: PEPTIC ULCER**

Moderator:

Franz J. Ingelfinger, M.D.
Editor, New England Journal of Medicine

Panelists:

T. Wilkins Davis, M.D.
Associate Professor of Radiology, Howard University College of Medicine

Albert I. Mendeloff, M.D.
Associate Professor of Medicine, Johns Hopkins University School of Medicine

Robert M. Zollinger, M.D.
Professor of Surgery, Ohio State University College of Medicine; Chairman, Department of Surgery, State University Hospital, Columbus

3:15 Recess to Visit Exhibits

3:35 **GASTROINTESTINAL SERIES: HIATAL HERNIA**

Moderator:

Robert M. Zollinger, M.D.

Panelists:

T. Wilkins Davis, M.D.

Thomas B. Ferguson, M.D.
Associate Clinical Professor of Thoracic and Cardiovascular Surgery, Washington University (St. Louis) School of Medicine

Franz J. Ingelfinger, M.D.

FRIDAY MORNING, NOVEMBER 22

C. Willard Camalier, Jr., M.D., *Presiding*
Member, Committee on Arrangements, 1968 Scientific Assembly

9:00 **COMPARATIVE PERFORMANCE OF EQUIVALENT DRUGS**

Moderator:

C. Willard Camalier, Jr., M.D.
Clinical Associate Professor of Surgery, Georgetown University School of Medicine

Panelists:

Gerhardt Levy, Pharm.D.
Professor and Chairman, Department of Pharmaceutics, School of Pharmacy, State University of New York at Buffalo

William Murray, M.D.
Assistant to Director for Drug Equivalency, Bureau of Medicine, Food and Drug Administration, Department of Health, Education, and Welfare

C. Joseph Stetler
President, Pharmaceutical Manufacturers Association

10:15 Recess to Visit Exhibits

10:35 **DRUG ABUSE: PROBLEMS AS VIEWED BY THE LAW ENFORCEMENT OFFICER, JUDICIARY, INVESTIGATOR, COMMUNITY HEALTH OFFICER, AND MEDICAL CLINICIAN**

Moderator:

Seymour Alpert, M.D.
Professor of Anesthesiology, George Washington University School of Medicine

Panelists:

Mr. Joseph Arpaio
Agent in Charge, Federal Bureau of Narcotics and Dangerous Drugs, Washington Field Office

The Honorable William B. Bryant
United States District Judge for the District of Columbia

Nathan Eddy, M.D.
Consultant on Narcotics and Drug Abuse, National Institutes of Health

Murray Grant, M.D.
Director of Public Health, District of Columbia

Sherman Kieffer, M.D.
Associate Director for Patient Care, National Institute of Mental Health

11:50 Recess for Lunch—Visit Exhibits

Commemorative Vases Available

An Opportunity to purchase the Medical Society's beautifully engraved glass commemorative vases, suitable for home and office adornment or for gifts to colleagues and friends, will be provided during the Assembly.

FRIDAY AFTERNOON, NOVEMBER 22

John M. Keshishian, M.D., *Presiding*
Member, Committee on Arrangements, 1968 Scientific Assembly

2:00 **ORGAN TRANSPLANTATION: THE HEART, KIDNEY AND LIVER**

J. Hartwell Harrison, M.D.
Elliot Carr Cutler Professor of Surgery, Harvard Medical School

Norman E. Shumway, Jr., M.D.
Professor of Surgery, Stanford University School of Medicine

Thomas E. Starzl, M.D.
Professor of Surgery, University of Colorado School of Medicine

3:15 Recess to Visit Exhibits

3:35 **ORGAN TRANSPLANTATION—PANEL DISCUSSION**

Moderator:

J. Hartwell Harrison, M.D.

Panelists:

Robert Schwartz, M.D.
Chief, Clinical Immunology Service, New England Medical Center Hospitals, Boston

Thomas E. Starzl, M.D.

Norman E. Shumway, Jr., M.D.

Acknowledgment: The Committee on Arrangements wishes to acknowledge the generous contributions of Eli Lilly and Company, The Upjohn Company, and the American Cancer Society, D. C. Division, which made this program possible.

OPEN TO THE PUBLIC

FRIDAY, NOVEMBER 22, 7:30 P.M.—INTERNATIONAL BALLROOM EAST

MORAL, RELIGIOUS AND LEGAL ASPECTS OF ORGAN TRANSPLANTATIONS

Welcome by **William S. McCune, M.D.**
President, The Medical Society of the District of Columbia

Moderator: **Mr. William Hines**
Washington Editor, World Book Science Service, Washington

Panelists:

The Reverend Dr. Joseph Fletcher
Professor of Social Ethics, Episcopal Theological School, Cambridge, Mass.

Paul D. Cantor, M.D., LL.B.
Adjunct Professor of Law and Professor of Medical Jurisprudence, Georgetown University

Norman E. Shumway, Jr., M.D.
Professor of Surgery, Stanford University School of Medicine

SATURDAY MORNING, NOVEMBER 23

Robert F. Donohoe, M.D., *Presiding*

Member, Committee on Arrangements, 1968 Scientific Assembly

9:00 I. CURRENT CONCEPTS OF RESUSCITATION

RESUSCITATION: WHAT IS IT?
Donald Benson, M.D.
Chairman, Department of Anesthesiology, Johns Hopkins University Hospital

ORGANIZATIONS WITHIN THE HOSPITAL
Arthur Peschin, M.D.
Department of Anesthesiology, George Washington University Hospital

I. PHASE-SHIFT INTRA-AORTIC BALLOON PUMPING IN CARDIOGENIC SHOCK
Major William Yahr, USAF, MC
Department of Surgery, Malcolm Grow Hospital, Andrews Air Force Base, Washington

II. MOVIE—PRESCRIPTION FOR LIFE
American Heart Association

III. PANEL DISCUSSION—THE STATUS OF RESUSCITATION

Moderator:

Patricia H. Russell, M.D.
Associate Professor of Anesthesiology, George Washington University Hospital

Panelists:

Donald Benson, M.D.
Arthur Peschin, M.D.
Major William Yahr, USAF, MC

The Scientific Exhibit

All Exhibits Open Thursday and Friday, 9 A.M. to 5 P.M.;
Saturday, 9 A.M. to 12 Noon

NEW CONCEPTS IN KIDNEY PATHOLOGY
V. J. Dardin, M.D., McLean, Va.

DRUG-INDUCED EMBRYOPATHY
Cecil B. Jacobson, M.D., Department of Obstetrics and Gynecology, and Ira G. Wong and Luke Terry, Cytogenetic Laboratory, George Washington University Hospital

GLAUCOMA, THE DISEASE THAT DESTROYS SIGHT
Claude L. Cowan, M.D., Chief, Division of Ophthalmology, Howard University College of Medicine

SURGICAL AND OBSTETRICAL COMPLICATIONS OF THE I.U.D.
David H. Kushner, M.D., Charles E. Townsend, M.D., Martin Thomas, M.D., and William J. Jaffurs, M.D., Medical Staff, Columbia Hospital for Women, Washington

CALCIFIED LIVER METASTASES
Albert J. Miele, M.D., X-ray Department, Sibley Memorial Hospital, Washington

THE MULTIPLE ETIOLOGIES AND THE CLASSIC ROENTGEN FEATURES OF PULMONARY EDEMA
Gerald J. Gelford, M.D., George Grames, M.D., and R. E. Blom, R. T., Washington Sanitarium and Hospital, Takoma Park, Md.

MEASURING ERYTHROCYTE, PLASMA AND EXTRACELLULAR FLUID VOLUME
Solomon N. Albert, M.D., Jo Shibuya, M.D., A. L. Smith, M.D., E. Varrone, M.D., and Chalom A. Albert, M.D., Department of Anesthesiology, Washington Hospital Center, Washington

CYSTIC FIBROSIS: ITS MANIFESTATIONS, DIAGNOSIS AND TREATMENT
Lucas L. Kulczycki, M.D., Director, Cystic Fibrosis Center, Children's Hospital of the District of Columbia

GYNECOLOGIC CANCER DETECTION IN HIGH-RISK PATIENTS
William J. Jaffurs, M.D., John Marlow, M.D., and Luis Arias-Bernal, M.D., Columbia Hospital for Women, Washington

CENTRAL MONITORING: A VALUABLE ADJUNCT TO PATIENT CARE
Patricia H. Russell, M.D., Charles S. Coakley, M.D., Cesar A. Caceres, M.D., and Jeannette Kuhn, R.N., Department of Anesthesiology, George Washington University Hospital

IN AND OUT SURGERY
Marie-Louise Levy, M.D., and Charles S. Coakley, M.D., Department of Anesthesiology, George Washington University Hospital

QUALITY CONTROL—A LABORATORY NECESSITY
Oscar B. Hunter, Jr., M.D., Washington

MANAGEMENT OF LEG CRAMPS OF PERIPHERAL VASCULAR DISORDERS
Francis J. Stern, M.D., Philadelphia

MAMMARY CANCER: DIAGNOSIS AND THERAPY
Jeanne C. Bateman, M.D., Alexandria, Va.

DISTRICT OF COLUMBIA POLITICAL ACTION COMMITTEE
Chad P. Combs, Field Representative, American Medical Political Action Committee, Chicago, and William H. Cooper, M.D., Chairman, District of Columbia Political Action Committee

INTERNATIONAL HEALTH SERVICES CLINIC: GEORGETOWN UNIVERSITY
James E. Banta, M.D., and Patrick J. Doyle, M.D., Georgetown University Medical Center

COMPONENT BLOOD THERAPY MEANS BETTER PATIENT CARE
John A. Shively, M.D., President, American Association of Blood Banks and U.S. Public Health Cancer Control Institute, Chicago

THE MODIFIED ANGLE SHOT IN BARIUM ENEMA EXAMINATION OF THE COLON
Louis Wener, M.D., Radiologist-in-Chief, Morris Cafritz Memorial Hospital, Washington

CLINICAL APPLICATION OF ANGIOGRAPHY
Louis Wener, M.D., Radiologist-in-Chief, Morris Cafritz Memorial Hospital, Washington

PROTECTION FROM X-RAYS
Milton Wiedler, District of Columbia Department of Public Health

RECOGNITION AND TREATMENT OF HIATUS HERNIA
Howard C. Baron, M.D., and Leon Sasson, M.D., Departments of Surgery and Gastroenterology, Jewish Memorial Hospital, New York

The Technical Exhibit

All Exhibits Open Thursday and Friday, 9 A.M. to 5 P.M.;
Saturday, 9 A.M. to 12 Noon

The following companies have contracted for space in the Technical Exhibit. The Assembly is made possible through the financial assistance of these exhibitors. They have items of interest to display and to discuss with you. A visit to each and every booth is suggested.

ABBOTT LABORATORIES
North Chicago, Illinois

ALAN G. DAY
Baltimore, Maryland

AMERICAN CANCER SOCIETY—DISTRICT OF COLUMBIA DIVISION, INC.
Washington, D.C.

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KLOMAN SURGICAL SUPPLY COMPANY
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MEDICAL-DENTAL BUREAU RESEARCH FOUNDATION
Washington, D.C.

MERCK SHARP & DOHME
West Point, Pennsylvania

PEOPLES DRUG STORES
Washington, D.C.

WM. P. POYTHRESS & CO., INC.
Richmond, Virginia

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Broomall, Pennsylvania

REYNOLDS & COMPANY
Washington, D.C.

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New York, New York

SANDOZ PHARMACEUTICALS
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W. B. SAUNDERS COMPANY
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E. R. SQUIBB, INC.
New York, New York

STUART DIVISION (ATLAS CHEMICAL)
Pasadena, California

SYNTEX LABORATORIES
Palo Alto, California

UNITED MEDICAL LABORATORIES, INC.
Portland, Oregon

Social Affairs

MEDICAL SOCIETY ANNUAL MEMBERSHIP LUNCHEON

Thursday, November 21, 1968—12:15 p.m.—International Ballroom East
GUEST SPEAKERS: **Frank Harden and Jackson Weaver**
WMAL Radio Program—"Harden and Weaver"
Free tickets (for 300 members) available at Advance Registration* or Assembly Registration Desk

DOCTORS' OFFICE STAFF DINNER

Thursday, November 21, 1968—6:30 p.m.—International Ballroom East
Entertainment by "Schuhplattler und Gebirgstrachten Verein Washingtonia" (Bavarian Dance Club)
Tickets ($9.50) available at Advance Registration or Assembly Registration Desk
Cocktails for this occasion courtesy of RUSSELL, MARSH & KENNEDY, INC.

WOMAN'S AUXILIARY LUNCHEON

Friday, November 22, 1968—12:15 p.m.—Crystal Room
GUEST SPEAKER: **The Honorable Walter E. Washington**
Commissioner, Washington, D.C.
Tickets ($7.00) available at Advance Registration or Assembly Registration Desk

GEORGETOWN UNIVERSITY MEDICAL ALUMNI LUNCHEON

Friday, November 22, 1968—12:15 p.m.—Jefferson Room
GUEST SPEAKER: To be announced
Tickets ($5.00) available at Medical Alumni Office or Assembly Registration Desk

GEORGE WASHINGTON UNIVERSITY MEDICAL ALUMNI LUNCHEON

Friday, November 22, 1968—12:15 p.m.—International Ballroom East
GUEST SPEAKER: **Harry F. Dowling, M.D.**
Professor and Head, Department of Medicine, University of Illinois College of Medicine
Tickets ($6.50) available at Medical Alumni Office or Assembly Registration Desk

ANNUAL RECEPTION AND DINNER DANCE

Saturday evening, November 23, 1968—6:30 p.m.—International Ballroom
Cocktails at 6:30 p.m.—Dinner at 8:00 p.m.
Dancing to the Music of MEYER DAVIS ORCHESTRA
Tickets ($12.50 per person) available at Advance Registration or Assembly Registration Desk
Cocktail Party—Courtesy of PEOPLES DRUG STORES
Music—Courtesy of BURTON, PARSONS & COMPANY, INC.
The Medical Society is most indebted to these organizations for contributing generously again to this gala event.

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* Advance Registration will be held at the Medical Society's Offices, November 18, 19 and 20, from 8:30 a.m. to 4:45 p.m.

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Head Start Health Programs*

ROLAND B. SCOTT, M.D.,† AND ALTHEA B. KESSLER, M.D.‡

THE HEAD START Child Development Program for preschool children of low-income families is now in its fourth year of operation. Grants are made by the Office of Economic Opportunity to local community action agencies. Operation of a Head Start center may be delegated by these agencies to a public or private nonprofit organization which has the capacity to operate such a program. Both summer and full-year programs for preschool children 3 years of age or older are funded.

Each local action group is required to name as medical director a local physician who is familiar with available health facilities and able to enlist the coöperation of the local medical community.

The Head Start Health Program is intended to provide continuous health care and supervision. Existing local health programs are to be used if available. It is expected that the local agency will provide health screening for all children enrolled and the following services for those who require them: immunization, outpatient treatment of acute and chronic illness, dental care, eye examination and glasses, speech and hearing examinations, and psychological evaluation and counseling.[1] Each child is examined as soon as possible after enrollment so that he may be in optimum condition to take advantage of the educational opportunities offered.

The initial screening procedure includes medical history, physical examination, visual screening, audiometry, dental examination, tuberculin test, urinalysis, and hemoglobin or hematocrit. Contracts for the screening procedure may be made with hospitals, clinics, private physicians, or dentists. In some communities this has been done on a voluntary basis, but usually a fee is provided. Further diagnostic procedures and follow-up are more expensive and more difficult to budget in advance. For this reason, help is usually sought from health departments, hospitals, clinics, religious organizations, foundations for a particular disease, and other voluntary agencies.

The following tables indicate the type of medical problems one might expect to see in disadvantaged preschool children. Table 1 concerns 223 Negro children screened in a Head Start Program in Washington, D. C.[2]

TABLE 1: Medical Problems Seen in 223 Head Start Children

Systemic Diseases Identified	No. Children
Central nervous system	2
Mental retardation	2
Eye defects	20
Ear, nose and throat	8
Cardiovascular system	3
Gastrointestinal (including hernia)	7
Genitourinary	2
Musculoskeletal	3
Hematologic	13
Endocrine and metabolic	4
Dermatologic	4
Allergic	3

Table 2 summarizes the medical experience in 80 per cent of all Head Start Programs nationwide in the summer of 1967.[3]

TABLE 2: Incidence of Selected Medical Problems

Condition	Per cent
Anemia	6.2
Mental retardation or learning problem	2.6
Skin disease	2.4
Heart murmur requiring specialist evaluation	1.4
Bronchial asthma	0.8
Orthopedic problem	0.45
Positive tuberculin test	0.45
Inguinal or femoral hernia	0.3
Needed measles immunization	43.0
Failed visual screening test	8.0
Dental caries	61.0
Other problems requiring further evaluation	17.0

There has been great variability in the adequacy of these health programs depending upon the availability of medical resources and the interest of local physicians and dentists.

In the fall of 1967 the American Academy of Pediatrics signed a contract with the Office of Economic Opportunity to provide medical care for the Head Start Program. The aim is to assign a consultant to each project to provide supervision on a year-round basis. The consultant becomes familiar with local problems and resources. In addition to making occasional on-the-spot evaluations he assists in planning the program and is available for advice throughout the school year.

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* Submitted for publication by the D. C. Chapter of the American Academy of Pediatrics, William A. Howard, M.D., Chairman, as a service to physicians of the Washington area.

† Professor and Chairman, Department of Pediatrics, Howard University College of Medicine; Chief Pediatrician, Freedmen's Hospital, Washington, D. C. 20001.

‡ Professor of Pediatrics, Howard University College of Medicine.

A regional Head Start Orientation session for consultants from Maryland, Delaware, District of Columbia, Virginia and Kentucky was held in Washington, D. C. on January 24, 1968. At that time pediatricians were assigned to specific project geographic areas. Pediatricians in the Washington Metropolitan Area who have received assignments are Drs. Robert J. Clayton, Melvin E. Jenkins, Roland B. Scott, Charles E. Hollerman, Joseph A. Bellanti, James Stallings, Jr., Robert H. Anderson, Harold McC. Hobart, Harriette Clark Chambliss, David M. French, and J. William Oberman.

The local community action agency in Washington is the United Planning Organization. The UPO or the organizations operating Head Start Programs funded through UPO have contracts with the following organizations for follow-up care for Head Start children: D. C. Health Department, Children's Hospital, George Washington Hospital, D. C. General Hospital, and Freedmen's Hospital.

At the present time there are a number of organizations in the Washington, D. C. area interested and active in providing health services for preschool children. This has resulted in considerable fragmentation of effort and service. There is need for greater emphasis on integration among medical, health, and welfare agencies in order to provide a more comprehensive delivery of service in view of existing manpower shortages. The medical profession can provide guidance and leadership in meeting this current challenge.

References

1. Community Action Program: An Invitation to Help Head Start Child Development Programs. Washington, D. C.: Office of Economic Opportunity.
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Narcotic Don'ts for the Physician

DON'T treat an ambulatory case of addiction. Addicts must be under proper control.
—Addicts go to several physicians at a time and receive duplicate drugs.

DON'T dispense any narcotics without keeping records.
—Bedside and office administration are permitted without record, but records can be helpful.

DON'T order your office narcotic needs on patients' prescription blanks.
—The law requires you to use an official order form.

DON'T resent a pharmacist's call for information about a prescription you may have written.
—The pharmacist is held responsible for filling forgeries. Please coöperate.

DON'T hesitate to call the Narcotic Section, Metropolitan Police Department. The number is 626–2493.
—Problems in narcotic drug abuse do arise. Your queries will be held as confidential as possible.

HEALTH DEPARTMENT NOTES

Cardiopulmonary Angiographic Laboratory

A NEW Cardiopulmonary Angiographic Laboratory which provides the heart patient with the most modern diagnostic equipment available was installed recently in D. C. General Hospital. Utilizing 2 closed-circuit X-ray televisions and other sophisticated equipment, a team of cardiologists is now able to observe the heart and great vessels in motion.

The prime purpose of the new laboratory is to assist in the diagnosis of anatomic or physiological disturbances of the heart and blood vessels in order to help guide the treatment of such illnesses.

The procedures performed in the laboratory require that the vital signs of the patient be constantly monitored. A new multichannel oscilloscope provides for continuous monitoring and makes available to the cardiologists valuable information concerning cardiac problems.

During the cardiac examination, various cathe-

Gotta make a pit stop to take my cough syrup.
Full speed ahead, Fred. These solid Cough Calmers can control that cough for 6 to 8 hours.
2

Robitussin-DM
Cough Calmers

Each Cough Calmer™ contains the same active ingredients as a half-teaspoonful of Robitussin-DM®. Glyceryl guaiacolate, 50 mg., Dextromethorphan hydrobromide, 7.5 mg. A. H. Robins Company, Richmond, Virginia 23220

A-H-ROBINS

ters are passed through the blood vessels to suspected areas of disease, at which time recordings are made. In some instances X-ray dye is injected through the catheters and several X-ray films are exposed. These diagnostic methods are extremely accurate, and the cardiologist may then recommend surgery or therapy.

A new rapid X-ray film changer, capable of taking 60 full-size X-ray films in 5 seconds, enables the cardiologist to observe contours, size, and position of many lesions of the individual chambers of the heart. In addition, an X-ray moving picture apparatus capable of taking 60 frames per second is available for use by the cardiology team to assist in the diagnosis of coronary diseases.

The results of various other laboratory procedures, such as blood analysis, exercise tests, pulmonary function studies, and other related data, are also available to the cardiologist in evaluating the condition of the heart patient.

Last year a $14 million construction and remodeling program was completed at D. C. General Hospital. This joined together the 3 main medical and surgical buildings. One of our purposes has been to modernize the supportive diagnostic and treatment facilities. The new Cardiopulmonary Angiographic Laboratory is a notable example of this modernization program.

Murray Grant, M.D., D.P.H.
D. C. Director of Public Health

►DOCPAC BRIEFS

or

The Estimates Were Too Low

Federal officials now feign surprise at the high cost of Medicare. As far back as 1964 officials of the Department of Health, Education, and Welfare were warned that the Government's cost estimates for Medicare would be exceeded in expenditure by at least 300 per cent, but no one paid attention.

To help get the Medicare bill passed, Health and Welfare officials told Congress that the Medicare law would cost only 1.3 billion dollars by 1970. Even though the benefits were subsequently restricted to make it less costly, the Government now admits that the cost will run this year 4.1 billion dollars.

When several organizations, including the AMA, warned of the future high costs, the Social Security Chief Actuary wrote an irate letter denying that the Government's estimates were wrong or that they were used to influence passage of the law. These phony estimates are typical of how potentially expensive welfare programs can be slipped through Congress.

Next month, on November 5, national elections will be held. Your Political Action Committee (DOCPAC) has helped to elect and reëlect several members to the House and is helping several Senators in their current campaigns. Other monies, collected through DOCPAC, have been allocated to campaign funds in key Senate and Congressional races all over the United States. They will aid members, both Republican and Democrat, who are friends of medicine and who are knowledgeable in the field of health problems and health legislation.

If you have not yet joined DOCPAC, your Medical Political Action Committee, please do.* And secondly, don't forget to vote on November 5. This is every citizen's responsibility.

William H. Cooper, M.D.
Chairman, D. C. Political
Action Committee

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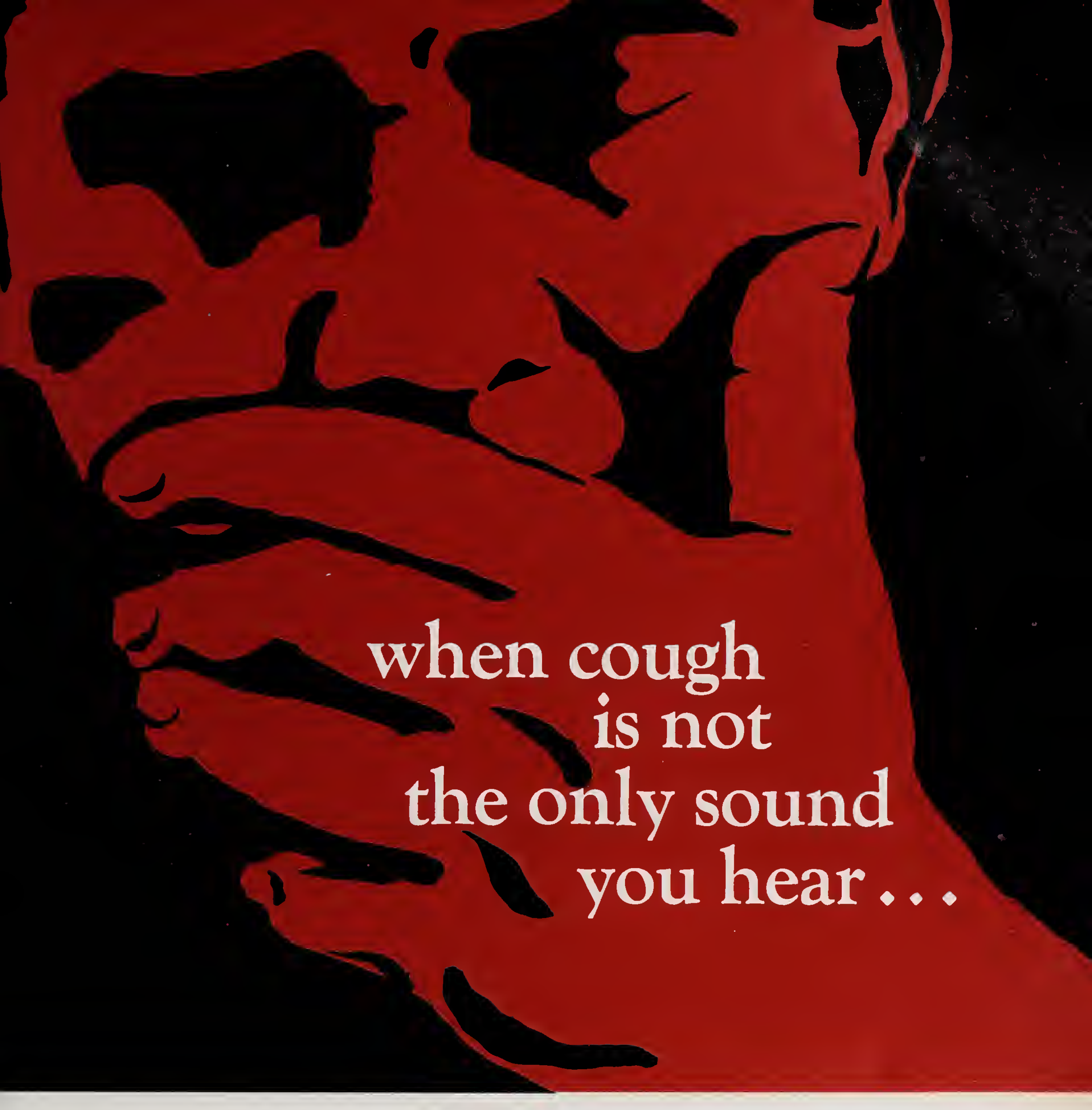
*Dues: Individual, $20.00.
Family, $30.00.



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. . . because OMNI-TUSS is indicated for cough associated with upper respiratory tract infections, bronchitis, bronchiectasis, bronchial asthma, emphysema, sinusitis and rhinitis, hay fever, or other allergic conditions. Any of these conditions may exhibit the general symptom syndrome—coughing, wheezing, bronchospasm, and tenacious mucus—which may benefit from the antitussive, bronchodilative, antihistaminic, and expectorant action of Omni-Tuss.

The therapeutic usefulness of Omni-Tuss is enhanced by a unique resin complex formulation providing the clinically desirable advantages of: (1) uniform drug availability throughout an extended period, (2) 8 to 12 hours of symptomatic control, (3) minimal dosage requirement, (4) minimal side effects.

Economical, efficient **b.i.d.** dosage—extremely well-tolerated by children, 6-12, and adults.

'Omni Tuss' Suspension: Each teaspoonful (5 cc.) contains 10 mg. codeine (Warning: May be habit-forming), 5 mg. phenyltoloxamine, 3 mg. chlorpheniramine, 25 mg. ephedrine, all as cation exchange resin complexes of sulfonated polystyrene, and 20 mg. guaiacol carbonate.

Available on prescription only. Class X exempt narcotic. Permissible on oral prescription.

Dosage: *Adults:* 1 teaspoonful (5 cc.) q12h.
Children (6-12 *years*): ½ teaspoonful q12h.

Side Effects: Minimal, but when encountered may include jitteriness, nausea, drying of mouth, insomnia, constipation, which disappear upon adjustment of the dose or discontinuance of treatment.

Precautions and Contraindications: For complete detailed information, refer to package insert or official brochure.

Strasenburgh
Strasenburgh Laboratories Division
Wallace & Tiernan Inc., Rochester, N.Y.

In the complex picture of moderate to severe anxiety...

there is a new reason for prescribing Mellaril (Thioridazine HCl)

effectiveness in mixed anxiety-depression

Long recognized for its usefulness in the treatment of moderate to severe anxiety, Mellaril is now also known to be effective against mixed anxiety-depression.

Often the symptoms of anxiety states are difficult to sort out—even with the most careful probing. The patient may manifest symptoms of agitation, restlessness, insomnia, somatic complaints. But what of the depression that may be mixed in the total picture? It is reassuring to know that Mellaril may be prescribed—with strong possibilities of success—when there is anxiety alone or a mixture of anxiety and depression.

Before prescribing or administering, see Sandoz literature for full product information, including adverse reactions reported with phenothiazines. The following is a brief precautionary statement.

Contraindications: Severe central nervous system depression, comatose states from any cause, hypertensive or hypotensive heart disease of extreme degree.

Warnings: Administer cautiously to patients who have previously exhibited a hypersensitivity reaction (e.g., blood dyscrasias, jaundice) to phenothiazines. Phenothiazines are capable of potentiating central nervous system depressants (e.g., anesthetics, opiates, alcohol, etc.) as well as atropine and phosphorus insecticides. During pregnancy, administer only when necessary.

Precautions: There have been infrequent reports of leukopenia and/or agranulocytosis and convulsive seizures. In epileptic patients, anticonvulsant medication should also be maintained. Pigmentary retinopathy may be avoided by remaining within the recommended limits of dosage. Administer cautiously to patients participating in activities requiring complete mental alertness (e.g., driving). Orthostatic hypotension is more common in females than in males. Do not use epinephrine in treating drug-induced hypotension. Daily doses in excess of 300 mg. should be used only in severe neuropsychiatric conditions.

Adverse Reactions: *Central Nervous System—* Drowsiness, especially with large doses, early in treatment; infrequently, pseudoparkinsonism and other extrapyramidal symptoms; nocturnal confusion, hyperactivity, lethargy, psychotic reactions, restlessness, and headache. *Autonomic Nervous System—*Dryness of mouth, blurred vision, constipation, nausea, vomiting, diarrhea, nasal stuffiness, and pallor. *Endocrine System—* Galactorrhea, breast engorgement, amenorrhea, inhibition of ejaculation, and peripheral edema. *Skin—*Dermatitis and skin eruptions of the urticarial type, photosensitivity. *Cardiovascular System—* Changes in the terminal portion of the electrocardiogram have been observed in some patients receiving the phenothiazine tranquilizers, including Mellaril (thioridazine hydrochloride). While there is no evidence at present that these changes are in any way precursors of any significant disturbance of cardiac rhythm, several sudden and unexpected deaths apparently due to cardiac arrest have occurred in patients previously showing electrocardiographic changes. The use of periodic electrocardiograms has been proposed but would appear to be of questionable value as a predictive device. *Other—*A single case described as parotid swelling.

Mellaril®
(Thioridazine HCl)
25 mg. t.i.d.

for moderate to severe anxiety and mixed anxiety-depression

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"The inconvenience of a cold"

For a cold, NTz® Nasal Spray provides rapid relief of nasal symptoms. Relief starts with the first spray which opens the inferior part of the common meatus. A second spray, a few minutes later, will shrink the turbinates to help provide sinus drainage and ventilation. Dosage may be repeated every three or four hours as needed, for temporary relief of symptoms. NTz is well tolerated but overdosage should be avoided.

As a sinusitis deterrent, NTz Nasal Spray can be used to keep the nasal passages open during a cold to help prevent development of acute sinusitis—or to help prevent the acute condition from becoming chronic.

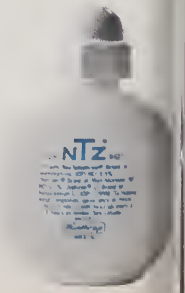
Supplied: NTz Nasal Spray, plastic squeeze bottles of 20 ml.; NTz Nasal Solution, bottles of 30 ml. (1 fl. oz.) with dropper.

NTz is more than a simple vasoconstrictor. It contains

Neo-Synephrine® (brand of phenylephrine) HCl 0.5 per cent, the major component, virtually synonymous with fast, efficient but gentle nasal vasoconstriction.

Thenfadil® (brand of thenyldiamine) HCl 0.1 per cent, topical antihistamine for reduction of rhinorrhea, sneezing or itching. It combats the allergic reactions that may occur in colds or sinusitis.

Zephiran® (brand of benzalkonium, as chloride, refined) 1:5000, antiseptic preservative and wetting agent to promote penetration and spread of the formula.



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News & Personals

Summary of Executive Board Actions

Proceedings of August 26 Meeting

Dinner for the Board members was served in the Exhibit Hall of the Medical Society. Dr. Edward W. Nicklas, Chairman, presided at the meeting which was attended by Drs. William S. McCune, Frank S. Bacon, Jed W. Pearson, Jr., Gudrun Augustin-Rueckert, Paul R. Wilner, Henry D. Ecker, Robert J. Coffey, John J. Hughes, and Mr. Thomas Sadler, Secretary. The meeting was called to order by Dr. Nicklas at 7:00 p.m.

The minutes of the July 22, 1968 Board meeting were approved with minor changes.

Committee Meetings: The Secretary reported the following committees had met since the last meeting of the Board:

July	23	Committee on Constitution and By-laws
	24	Membership Committee
	25	Committee on Rehabilitation
	30	Committee on Medical Ethics
August	2	Subcommittee on Scientific Exhibit, Annual Scientific Assembly
	13	Grievance Committee
	22	Finance Committee

Report to the Board on Regional Medical Program: By invitation of the Executive Board, Dr. Thomas W. Mattingly, Coördinator, Regional Medical Program, appeared to present his final report on RMP activities together with his recommendations to the grantee. At the conclusion of his report Dr. Nicklas thanked him for his excellent presentation and asked that he submit his recommendations in writing. Dr. McCune praised Dr. Mattingly for the fine contribution he has made to RMP. In turn Dr. Mattingly thanked the Board for inviting him and retired.

Society's Councils: Considerable discussion was held regarding the functioning of the Society's Councils as spelled out in Chapter IX of the By-laws. It was agreed that the premise of the Councils is fine but implementation has not been practical. Dr. Bacon requested guidance of the Board in this matter, since he is well under way in appointing his committees for 1969. A motion was made and carried that this matter be referred to the Committee on Constitution and By-laws and that it consider deleting all references to councils from the By-laws.

Election of Nominating Committee: Dr. McCune presented a list of 21 physicians whom he had chosen to appear on the ballot for the Nominating Committee. After review, the Board approved the list.

Luncheon Meeting with Dr. Grant: Dr. McCune summarized the items discussed with Dr. Grant at their August 26 luncheon meeting. He stated that Dr. Grant would in general support the proposed amendments to the Healing Arts Practice Act. Dr. Grant suggested that an attorney be on the Commission. He also agreed that he would have the proposed amendments rewritten in what he termed "proper legal terminology."

Dr. Grant reported that publicity pertaining to Medicaid and the hospitals was not correct; that hospitals will receive 100 per cent of the cost of inpatient care and 80 per cent of the cost of outpatient care.

Dr. Grant mentioned that he had been approached by a delegation from the Medical Committee for Human Rights to set up a clinic in Anacostia, with no restrictions on service, but indicated that he did not approve of such a proposal.

In reply to a query Dr. Grant emphasized that all available beds in D. C. General Hospital were being utilized.

Dr. McCune stated that he was still not fully satisfied with the discussion on neighborhood health centers. It was the consensus of the Board that Dr. Grant should be more responsive to the wishes of the Medical Society and that he himself should clarify many points regarding the neighborhood health centers. It was therefore agreed that Dr. Grant be invited to meet with the Board on September 23.

Membership Committee: The Board accepted the recommendation of the Membership Committee that a Dr. Philip Burka Memorial Citation and Award be established. Dr. Irving Burka had presented the proposal to the Committee and stated that further details would be supplied for its approval and recommendation to the Executive Board.

The Board approved the recommendation that, upon the death of a Society member, a notice, to include the seal of the Society, be placed in the obituary columns of all Washington newspapers.

Approval was also given to the recommendation of the Committee, with the concurrence of the Committee on Public Information, that the monthly Newsletter of the Society and the monthly Current Medical Events calendar be combined under the title, "News of the Medical Society of the District of Columbia."

Nominations for Society Awards: The Board approved the following recipients of awards, to be presented during the Scientific Assembly, as recommended by the Committee on Public Information: (a) Certificate for Meritorious Service to Dr. W. Montague Cobb; (b) the John Benjamin Nichols Award to Planned Parenthood of Metropolitan Washington, D. C., Inc.; and (c) Robins Community Service Award to Dr. E. Clarence Rice.

Change in Constitution and By-laws: Approval was given to amend Chapter V, Article I, Sections 1 and 5, as recommended by the Committee on Constitution and By-laws, in order to authorize the employment of an officer to act both as Executive Director and Secretary-Treasurer.

Corporate Practice of Medicine: Mr. Sadler reviewed, for informational purposes only, the opinion of the Medical Ethics Committee on the corporate practice of medicine. He stated that the Medicolegal Committee is exploring all facets of the corporate practice of medicine.

Other Business

1. A letter from Dr. Samuel Dove was reviewed. The Board concurred with Dr. Dove that listing physician specialties in the Yellow Pages of the Telephone Directory would possibly be more troublesome than helpful to all concerned. Accordingly, the Board voted to ask the Medical Bureau Committee to consider revising the Medical Bureau advertisement in the Yellow Pages in order to emphasize services available.

2. Dr. McCune discussed briefly the contents of a letter from Mr. Eugene W. Weigman regarding Adult Education Week. It was agreed that Dr. McCune advise Mr. Weigman that the Medical Society is very much interested in participating in Adult Education Week but the Society cannot contribute any funds.

3. Mr. Sadler read a letter from Mr. Warren E. Magee, the Society's legal counsel, in which Mr. Magee advised that "the House and Senate, in passing the District of Columbia tax bill, have excluded from that tax bill the attempted elimination of the exclusions in the present unincorporated business tax law of the District of Columbia, and also have refused to impose a surtax on nonresident physicians and other professional people" who have offices in the District of Columbia.

4. The Board agreed that Mr. Sadler should advise officials of the Washington Technical Institute that the Medical Society will be glad to participate in its functions whenever feasible and possible and that it will be pleased, as requested, to be represented on its Advisory Committee.

5. The Secretary announced that Community Health Week would be held October 20 through 26 and that specific details will be outlined at a later date.

There being no further business, the meeting was adjourned at 10:10 p.m.

MEETINGS

Georgetown University's Fourth Annual Symposium on Family Psychotherapy will be held at the Georgetown Medical Center, November 1 and 2. Further information may be secured by writing Dr. Francis D. Andres, 3710 Chanel Road, Annandale, Va. 22003.

Members of the Medical Society are asked to curtail their regular schedules for November 21, 22 and 23 so they will be able to attend the Society's **Annual Scientific Assembly** at the Hotel Washington Hilton. The scientific program, scientific and technical exhibits, and social affairs are published in this issue of the MEDICAL ANNALS. All physicians in the Washington Metropolitan Area as well as residents and interns are cordially invited to attend. There is no registration fee.

The American Medical Association will hold its **22nd Clinical Convention** in Miami Beach, Florida, December 1–4, at the Miami Beach Convention Hall. The scientific exhibits will be available for viewing from noon Sunday until the session ends on Wednesday afternoon. Postgraduate courses will be offered on diabetes, fluid and electrolyte balance, and thyroid disease. The Scientific Program for the Convention will be published in the October 21 issue of *The Journal of the American Medical Association.*

On Saturday, November 30, preceding the Clinical Meeting, the AMA will hold its **National Conference on Community Health Planning** at the Hilton Plaza Hotel. Case histories of health planning activities at the state and local levels will be presented during the morning session. The afternoon program will be devoted to multiple workshops designed to provide a thorough orientation to the actual process of community planning.

The Tenth National Conference on the Medical Aspects of Sports will convene at the Hotel Deauville on Sunday, December 1. The Conference is held annually in conjunction with and on the first day of the AMA Clinical Convention. Luncheon speaker will be Payton Jordan, Head Coach of the 1968 U. S. Olympic Track and Field Team, who will discuss "The Olympics in Retrospect." The Conference is open to nonmedical athletic personnel as well as interested physicians.

The Georgetown University Medical Center announces a **Course in Orthopedic Pathology**, sponsored by the Orthopedic Division, to be held at the Marriott Motel at Key Bridge, Washington, D. C., January 5–12, 1969. This will be an 80-hour course, including 30 hours of laboratory, under the direction of Dr. Lent C. Johnson of the Armed Forces Institute of Pathology. Further information may be secured from Dr. G. W. Hyatt, 3800 Reservoir Road, N.W., Washington, D. C. 20007.

GENERAL NEWS

Dr. Arthur Edwards Wentz, neurologist, has been appointed by the Executive Board of the Medical Society of the District of Columbia to succeed Dr. Thomas W. Mattingly as Coördinator of the Metropolitan Washington Regional Medical Program, which is implementing the President's Heart, Cancer, and Stroke Program. He assumed his new position, September 1.

Dr. Wentz, new Coördinator of RMP in the Metropolitan Washington Area

Dr. Wentz was appointed by the Federal Aviation Agency in 1959 to assist in the organization of a program of clinical research in the field of aviation medicine. This unique organization of government-sponsored scientists was integrated into the staff of, and with laboratories located in, the Georgetown University Medical Center. He continued as Director of the Georgetown Clinical Research Institute until 1966. From 1960 to 1966 he also served on the teaching staff of Georgetown University as Associate Clinical Pro-

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When she gets a bacterial infection, think of Tetrex-F.®

Like the debilitated or diabetic patient, she may be susceptible to monilial overgrowth.

That's why she needs the extra protection of Tetrex-F whenever tetracycline therapy is indicated. Tetrex-F provides well-tolerated tetracycline phosphate complex for the bacterial infection—nystatin to help prevent monilial overgrowth.

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(tetracycline phosphate complex-nystatin)

Whenever monilia may threaten tetracycline therapy

PRESCRIBING INFORMATION. For complete information consult Official Package Circular. *Indications:* Infections of respiratory, gastrointestinal and genitourinary tracts and skin and soft tissues due to tetracycline-sensitive organisms, in patients with increased susceptibility to monilial infections. *Contraindications:* The drug is contraindicated in patients hypersensitive to its components. *Warnings:* Photodynamic reactions have been produced by tetracyclines. Natural and artificial sunlight should be avoided during therapy. Stop treatment if skin discomfort occurs. With renal impairment, systemic accumulation and hepatotoxicity may occur. In this situation, lower doses should be used. Tooth staining and enamel hypoplasia may be induced during tooth development (last trimester of pregnancy, neonatal period and childhood). *Precautions:* Bacterial superinfections may occur. Infants may develop increased intracranial pressure with bulging fontanels. In gonorrheal therapy, serologic tests for syphilis should be conducted initially and monthly for 3 months. *Adverse Reactions:* Glossitis, stomatitis, nausea, diarrhea, flatulence, proctitis, vaginitis, dermatitis, and allergic reactions may occur. *Usual Adult Dosage:* 1 capsule q.i.d. Continue for 10 days in Beta-hemolytic streptococcal infections. Administer one hour before or two hours after meals. *Supplied:* Capsules, bottles of 16 and 100. Each capsule contains tetracycline phosphate complex equivalent to 250 mg. tetracycline HCl activity and 250,000 units of nystatin. For Oral Suspension, 125 mg. tetracycline and 125,000 u. nystatin/5 ml., 60 ml. bottles. **A. H. F. S. Category 8:12**

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fessor of Neurology. Since then he has been associated with the Food and Drug Administration as Extramural Research Advisor in the Office of the Associate Commissioner for Science.

Dr. Wentz, who was born in South Charleston, Ohio, received his medical degree from Ohio State University. During World War II he served in the Pacific and the Orient with the U. S. Navy Medical Corps and is presently a Captain in the Naval Medical Corps Reserve.

Dr. Robert Q. Marston, who has been Director of the Regional Medical Programs at the National Institutes of Health, has been appointed Director of NIH to succeed Dr. James A. Shannon, who retired, September 1, after heading the research center since 1955.

Dr. Marston had been Administrator of the Health Services and Mental Health Administration since April 1, a co-equal body with NIH in the Health, Education, and Welfare Department's tripartite Health Division.

Dr. Shannon has become special advisor to President Frederick Seitz of the National Academy of Sciences. During the 13 years of Dr. Shannon's administration, NIH experienced a period of unprecedented expansion in both the scope and diversity of its research programs and in the appropriations made available to it in support of these activities.

As of June 30, 1968, the Division of Regional Medical Programs severed its relations with the National Institutes of Health and was transferred to the newly created Health Services and Mental Health Administration. Dr. Stanley W. Olson, Program Coördinator of the Tennessee Mid-South Regional Medical Program, has succeeded Dr. Marston as Director of DRMP.

A District of Columbia Respiratory Care Directory has been compiled and is being distributed by the D. C. Tuberculosis and Respiratory Disease Association at 1714 Massachusetts Avenue, N.W. The Directory provides a handy inventory of respiratory care resources in the community.

Twenty hospitals from the District of Columbia and Montgomery and Prince George's Counties are listed along with their facilities, equipment, personnel, etc. available for respiratory care. Several hospitals were not included because the survey questionnaire was not considered applicable to them.

The Association acknowledges the efforts of Dr. Harold M. Silver, Chairman of its Respiratory Disease Committee, and Dr. Curt A. Ries, of the D. C. Department of Public Health, for assisting in the composition of the survey questionnaire.

Copies may be secured by calling the Association at 234-7100.

James E. Hicken, Ph.D., was named Director of Research in Medical Education at the Georgetown University School of Medicine, effective September 1. He will work with the School in developing and evaluating a new curriculum.

For the past 3 years the Medical School has been considering plans for updating the present curriculum to permit a more flexible course of study with greater student opportunity for contact with the community and for more individual research and study. As they are approved by the Executive Faculty, the changes will begin to take effect in September 1969.

Dr. Hicken came to Georgetown from Florida where he has been Professor of Education at the University of Miami and Co-director of the Florida Migratory Child Research Project.

"Drownproofing," a water-survival technique, is explained and illustrated on a wallet-sized card which may be obtained from the Public Health Service. Drownproofing is a technique for staying afloat, even in rough water, for a long period with a minimum exertion of energy. To obtain up to 100 wallet-sized cards free, address Public Inquiries, National Center for Urban and Industrial Health, 222 East Central Parkway, Cincinnati, Ohio 45202.

The Reverend Mark H. Bauer, S.J., has resigned as Vice President for Medical Center Affairs at Georgetown University but will remain in his position until a successor is named. Since becoming Vice President in 1963 he has helped to develop extensive expansion plans for Georgetown Hospital and the Schools of Medicine and Dentistry. Before assuming this post at the Medical Center, Father Bauer was Associate Professor of Biology and Research Associate in Physiology at Georgetown. He plans to return to teaching and research.

The Board of Directors at **Children's Hospital** has announced its intention to relocate the Hospital to a new site on the grounds of the Washington Hospital Center.

After the picnic even Gramps Was a victim of intestinal cramps

Parepectolin for quick relief of acute diarrhea
...soothes colicky pain with paregoric*
...consolidates fluid stools with pectin
...adsorbs irritants with kaolin,
and protects intestinal mucosa

In elderly patients it is particularly important to stop the diarrhea fast. Parepectolin helps you control diarrhea promptly and gain the patient's confidence until etiology has been determined.

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Each fluid ounce of creamy white suspension contains:
*Paregoric (equivalent) .... (1.0 dram) 3.7 ml.
Contains opium (¼ grain) 15 mg. per fluid ounce.
warning: may be habit forming
Pectin .... (2½ grains) 162 mg.
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(alcohol 0.69%)
Usual Adult Dose: One or two tablespoonfuls three times daily.

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PERSONALS

Dr. Thomas E. Curtin, who has served as Director of Medical Education at Providence Hospital since 1965, has been named to the new post of Medical Director of Providence. Dr. Curtin, who served both as intern and resident physician in medicine at Providence, was Chairman of the Department of Medicine from 1960 to 1967. He is Clinical Assistant Professor of Medicine at Georgetown University.

Dr. James W. Long has been appointed Associate Chief of the Physician Supply and Utilization Branch of the Division of Physician Manpower, Bureau of Health Manpower, National Institutes of Health. The year-old Division of Physician Manpower provides a Federal focus for programs dealing with problems of the education, supply, and utilization of physicians in the United States. The Physician Supply and Utilization Branch is concerned with the development and maintenance of valid information on physician supply, need, and distribution to provide estimates and projections of shortages and to make continuing recommendations regarding these resources.

Dr. Clifton R. Brooks has been appointed to the United States Air Force (Reserve) with the rank of Lt. Colonel, effective July 4. He will serve as the Flight Surgeon for the 27th Medical Service Squadron (Reserve) at the Langley Air Force Base in Virginia. Dr. Brooks served 19 years in the Naval Reserve and resigned his commission as Lt. Commander in 1961 in order to continue in the Air Force Reserve Program.

Dr. R. Frank Jones, Medical Director of Freedmen's Hospital, was elected Senior Vice President and designated Acting President of the Health and Welfare Council of the National Capital Area until December 31, 1968. He will serve in the latter capacity in order to permit Mr. Glenn E. Watts, President of the Council, to devote most of his time to conducting the 1968 United Givers Fund campaign.

Dr. Peter D. Comanduras, who co-founded MEDICO, the international medical aid agency, with the late Dr. Thomas Dooley, addressed the tenth anniversary dinner on June 6 at the New York Hilton. Dr. Comanduras, an internist of

Washington, was Secretary-General of MEDICO when it merged with CARE in 1962. He continued to head the medical program until 1963 when he resigned to resume his private practice. Now in charge of professional relations for Merck Sharp & Dohme International, he remains active on the MEDICO Advisory Board. He is an Associate member of the D. C. Medical Society.

A portrait of **Dr. Brian Blades,** Chairman of the Department of Surgery, George Washington University School of Medicine, hangs in the Blades Conference Room at the Hospital. Commissioned by the Airlie Foundation, it is the work of Washington artist Daniel B. Mistrik. Dr. Murdock Head, Director of the Airlie Foundation, unveiled the painting during a brief ceremony in June. Dr. Blades has been a member of the medical faculty at George Washington University since 1946.

OBITUARIES

Elma Bebee Carr, M.D.

(1888–1968)

Dr. Elma B. Carr, a Life member of the Medical Society of the District of Columbia, died, August 16, 1968 at the Washington Hospital Center. She lived and maintained her offices at Bellevue Hotel, 15 E Street, N.W.

Dr. Carr received her early education in Morganfield, Kentucky, where she was born, June 15, 1888. After coming to Washington in 1914 for employment in the Government she decided on a professional career. Enrolling at George Washington University, she received her A.B. degree in 1920, a Master's degree in 1921, and her medical degree in 1928. She interned at New England Hospital for Women and Children in Boston.

Dr. Carr began her general practice in Washington in 1929. She had staff privileges at most of the Washington hospitals.

Elected to Active membership in the Medical Society of the District of Columbia in 1934, she became a Life member in 1965. She was a member of the American Medical Association, the American Rheumatism Association, the George Washington University Medical Alumni Association, and a member and Past President of the D. C. Rheumatism Society.

Russel Jon Jansen, M.D.

(1899–1968)

Dr. Russel J. Jansen, obstetrician and gynecologist in Washington since 1926, died, July 23, 1968 at his home, 7066 Wyndale Street, N.W. He maintained offices at 1028 Connecticut Avenue, N.W. prior to his retirement, July 1.

Born in Red Wing, Minnesota, October 28, 1899, Dr. Jansen received his degree in medicine from George Washington University in 1926. He interned at Garfield Memorial Hospital the following year and continued on the staff for another year as resident physician. He trained in his chosen specialty at the Woman's Hospital in New York, from 1933 to 1935.

Dr. Jansen continued his long association with Garfield Hospital until and after it merged into the Washington Hospital Center. He was also on the staff of Doctors and Sibley Hospitals and Columbia Hospital for Women. He was on the teaching staff of George Washington University School of Medicine from 1940 to 1950.

Dr. Jansen was a Fellow of the American College of Surgeons and of the American College of Obstetricians and Gynecologists. He was elected to Active membership in the Medical Society of the District of Columbia in 1932. He was a member of the American Medical Association, the Southern Medical Association, and a member and Past President of the Washington Gynecological Society. He belonged to the Chevy Chase Club.

Besides his wife, Mrs. Helen Carter Jansen, he leaves a son, Jon Jansen, of Cumberland, Md., and a daughter, Mrs. Louise Monchick, of Chevy Chase, Md.

Charles F. Finley, M.D.

(1915–1968)

Dr. Charles F. Finley, an Associate member of the Medical Society of the District of Columbia, died, May 9, 1968, in Richmond, Va.

Dr. Finley was born in Mt. Morris, New York, July 22, 1915. He earned his baccalaureate degree from the University of Rochester in 1936 and his medical degree from George Washington University in 1939. He interned at Sibley Memorial Hospital the following year.

During World War II he was commissioned Captain in the Medical Corps of the U.S. Army and served with the 101st Evacuation Hospital in France and Germany.

Dr. Finley was elected to Active membership in

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the Medical Society of the District of Columbia in 1948. He transferred to Associate membership in 1956 following his departure from Washington to Fontana Dam in Graham County, North Carolina. He later practiced in Burlington and Fayetteville, N. C., before moving to Richmond.

His wife, Mrs. Dorothy B. Finley, 9811 Fernleigh Drive, Richmond, Va., survives him.

CV Response to Intracranial Pressure—Ducker et al

(*Continued from page 525*)

primary inotropic response on the myocardium,[5, 8] and increased peripheral resistance[3-6] as primary mechanisms in the pressor response elicited by increases in intracranial pressure. Our experimental studies indicate that all these mechanisms are involved and that each is recruited in turn in order to maintain systemic diastolic arterial pressures at levels sufficient to exceed the intracranial pressure. By gradations of sympathetic discharge, venoconstriction, a myocardial inotropic response, and peripheral arterial constriction are sequentially called into play in order to maintain brain perfusion.

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Chylothorax in Newborn—Said, Rosalinas and Reichelderfer

(*Continued from page 540*)

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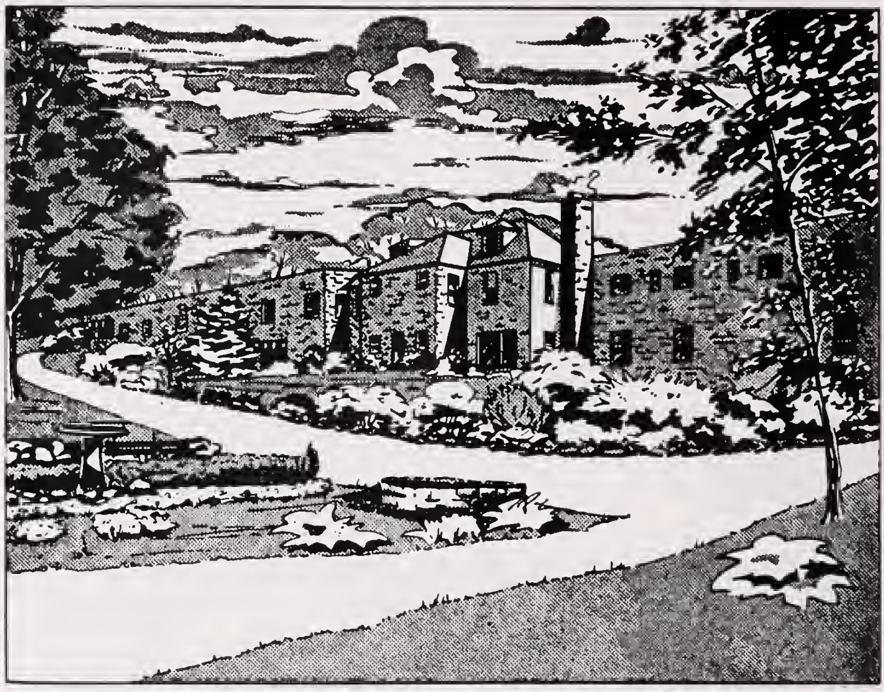

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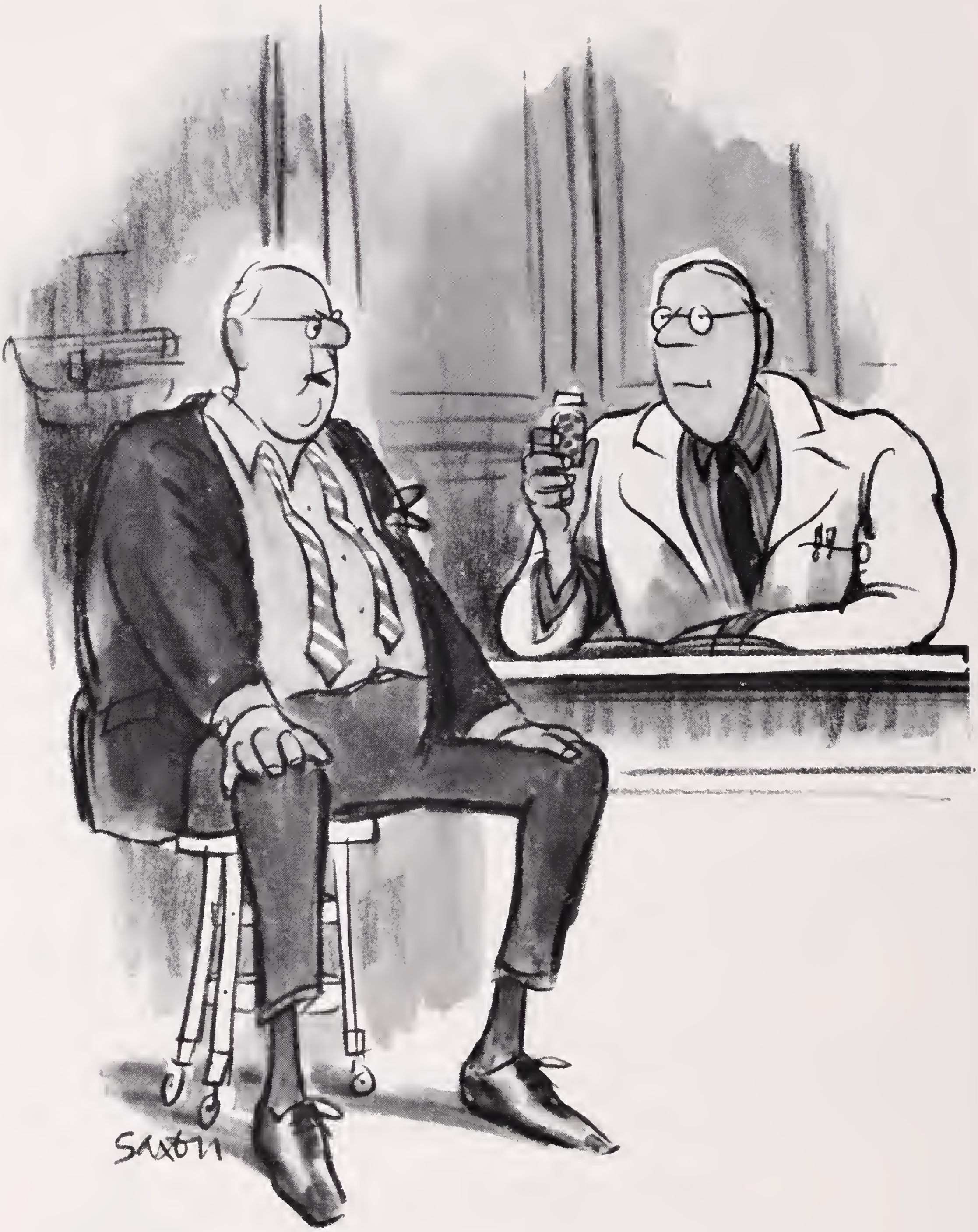

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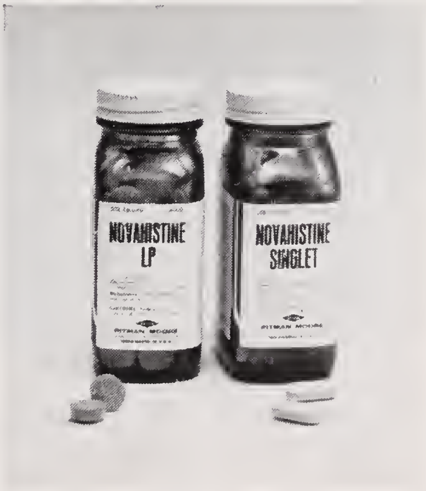

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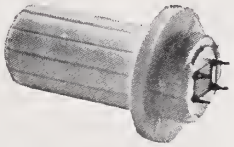

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Complete blood and platelet counts should be done prior to and at regular intervals during treatment with the drug (see recommendations under "Important Note and Precautions") to help in the early detection of serious bone marrow injury. Abnormalities in initial blood tests should rule out use of the drug. Also, patients should be made aware of such early toxic signs of a potential hematologic problem as fever, sore throat, mouth ulcers, easy bruising and petechial or purpuric hemorrhage. Should such signs appear, the patient should be advised to discontinue the drug and to report to the physician immediately.

Indication Tegretol is indicated in the treatment of the pain associated with true trigeminal neuralgia. It is not a simple analgesic and should never be administered for relief of trivial facial aches or pains.
Contraindication Do not use in those with a known sensitivity to any tricyclic compound or in those being treated with M.A.O.I. agents. As long a period as possible should elapse before using Tegretol in patients who have been treated with M.A.O.I.'s, with a minimum of 7 days. In such cases, initial dosage should be low and the patient's reaction to gradual increments closely observed.
Important Note and Precautions Familiarity with the clinical symptoms which lead to an accurate diagnosis of true trigeminal neuralgia and with the complete prescribing information, careful patient selection, a thorough examination before treatment and close patient supervision throughout the treatment period are essential to the safe and effective use of this drug.
Where feasible, Tegretol should not be used in conjunction with any potent drug which may increase the possibility of toxic reactions.
In pregnancy, the drug should not be prescribed during the first trimester and thereafter only to patients in whom the clinical situation warrants the potential risk. It should not be administered to nursing mothers.
Patients with increased intraocular pressure should be closely observed during treatment with this drug because of its anticholinergic effect.
Because of the drug's relationship to other tricyclic compounds, the possibility of activation of latent psychosis and, in the elderly, of confusion or agitation, should be considered.
Dizziness and drowsiness may occur and patients should be cautioned about the hazards of operating machinery or automobiles and of engaging in other hazardous tasks.
Use cautiously in patients with a history of coronary artery disease, organic heart disease, congestive failure or liver disease.
Before initiating therapy, the following laboratory procedures should be performed:
1. Complete blood and platelet counts which, if abnormal, should rule out the use of the drug.
2. Baseline evaluations of liver function.
During treatment with Tegretol, the following laboratory procedures should be performed:
1. Complete blood and platelet counts should be done at intervals of one week during the first month of drug treatment, every two weeks during the second and third months, and at monthly intervals thereafter for as long as the patient is taking the drug. A trend toward a decreasing white blood cell count should suggest a dosage reduction and more frequent laboratory and clinical evaluations. Should this trend continue, the drug should be discontinued.
2. Liver function tests must be performed at regular intervals during trea ment with this drug since liver damage may occur during therapy. The drug should be discontinued immediately in cases of aggravated liver dysfunction or active liver disease.
3. Periodic eye examinations, including slit-lamp, funduscopy and tonom- etry, are recommended for patients being treated with this drug since many phenothiazines and related drugs have been shown to cause eye changes.
4. Complete urinalysis and BUN should be done on patients treated wit Tegretol because of observed renal dysfunction.
Adverse Reactions Dizziness, drowsiness, unsteadiness on the feet, nausea, vomiting, aplastic anemia, transitory leukopenia, agranulocytosis, eosinophilia, leukocytosis, thrombocytopenia, purpura, abnormalities in liver function tests, cholestatic and hepatocellular jaundice, urinary frequency, acute urinary retention, oliguria with elevated blood pressure, albuminuria, glycosuria, elevated BUN, microscopic deposits in the urine impotence, disturbances of coordination, confusion, headache, fatigue, blurred vision, transient diplopia and oculomotor disturbances, speech disturbances, abnormal involuntary movements, peripheral neuritis and paresthesias, depression with agitation, talkativeness, nystagmus, tinnitus, paralysis and other symptoms of cerebral arterial insufficiency, pruritic and erythematous rashes, urticaria, Stevens-Johnson syndrome, photosensitivity reactions, alterations in skin pigmentation, exfoliative dermatitis, alopecia, diaphoresis, recurrence of thrombophlebitis, erythe multiforme and nodosum, aggravation of disseminated lupus erythematosus, gastric distress, abdominal pain, diarrhea, constipation, anorexia, dryness of the mouth and pharynx, glossitis, stomatitis, fever, chills, adenopathy, lymphadenopathy, aching joints and muscles, leg cramps, conjunctivitis, left ventricular failure, aggravation of hypertension, hypotension, syncope and collapse, edema, aggravation of coronary artery disease and congestive heart failure. (Whether these cardiovascular effe are drug-related is not known. However, some of these complications ha resulted in fatalities.) The necessity for discontinuing the drug should be dictated by the gravity and severity of the adverse reactions.
Dosage and Administration The drug should always be taken with meals, if possible.
Initial: One-half tablet (100 mg.) b.i.d. on the first day. Thereafter, the dos should be increased in one-half tablet (100 mg.) increments every 12 hou until freedom from pain is achieved. To relieve pain, between 200 mg. an 1200 mg. per 24 hours may be necessary.
Maintenance: Initial control of pain can be maintained in most patients with a dose of 400 mg. to 800 mg. daily. Maintenance doses may range between 200 mg. and 1200 mg. daily.
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Warning: Clinical reports have suggested that there may be a risk of teratogenesis associated with the use of this compound during the first trimester of pregnancy. Unless, in the opinion of the prescribing physician, the potential benefits outweigh the possible risks, it should not be used during the first trimester of pregnancy. Cardiovascular complications, including myocardial infarction and arrhythmias, have occasionally occurred in susceptible individuals. Patients with cardiovascular disease should be given the drug only under careful observation and in low dosage.
Precautions: Since suicide is always a possibility in severely depressed patients and one which may persist until significant remission occurs, such patients should be carefully supervised during early treatment. Some severely depressed patients may also require hospitalization and/or concomitant electroconvulsive therapy. Because of its anticholinergic effect, caution should be observed in prescribing the drug for patients with increased intraocular pressure.
In rare instances, transient cardiac arrhythmias have occurred in hyperthyroid patients and in patients receiving thyroid medication when this compound was added to the regimen. Imipramine may block the pharmacologic activity of guanethidine and other related adrenergic neuron-blocking agents.
The drug is not recommended at the present time in patients under 12 years of age.
Adverse Reactions: Dryness of the mouth, tachycardia, constipation, disturbances of accommodation, sweating, dizziness, weight gain, urinary frequency or retention, nausea and vomiting, peripheral neuritis, mild parkinson-like syndrome, tremors, rare cases of falling in elderly patients, confusional states (with such symptoms as hallucinations and disorientation), activation of psychosis in schizophrenics and agitation (including hypomanic and manic episodes) which may require dosage reduction and/or addition of a tranquilizer or temporary discontinuation of the drug, epileptiform seizures, orthostatic hypotension and substantial blood pressure fall in hypertensive patients, purpura, transient jaundice, bone marrow depression including agranulocytosis, sensitization and skin rash including photosensitization, eosinophilia, and mild withdrawal symptoms on sudden discontinuation after prolonged treatment with high doses. Occasional hormonal effects (impotence, decreased libido, and estrogenic effects) may be observed. Atropine-like effects may be more pronounced (e.g. paralytic ileus) in susceptible patients and in those using anticholinergic agents (including antiparkinsonism drugs).
Outpatient Adult Dosage: Initially, 75 mg. daily, increased, if necessary, to 150 or 200 mg. Maintenance dosage may be lower, 50 to 150 mg. daily, if possible.
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October 19–24: American Academy of Pediatrics. Palmer House, Chicago. Address: Dr. Robert G. Frazier, Executive Director, 1801 Hinman Avenue, Evanston, Ill. 60204.

October 20–23: Association of Military Surgeons of the United States. Sheraton Park Hotel, Washington. Address: Brig. General Frank E. Wilson, USAR (Ret.), 1500 Massachusetts Avenue, N.W., Washington, D. C. 20005.

October 27: American Association of Ophthalmology. Palmer House, Chicago. Address: Mr. Lawrence A. Zupan, Executive Secretary, 1100 17th Street, N.W., Washington, D. C. 20036.

October 27–30: American College of Gastroenterology. Hotel Statler-Hilton, Boston. Address: Mr. Daniel Weiss, Executive Director, 33 West 60th Street, New York, N. Y. 10023.

October 27–November 1: American Academy of Ophthalmology and Otolaryngology. Palmer House, Chicago. Address: Dr. William L. Benedict, Executive Secretary, 15 Second Street, S.W., Rochester, Minn. 55901.

October 28–31: American Association of Blood Banks. Shoreham Hotel, Washington. Address: Miss Lois J. James, Executive Secretary, 30 North Michigan Avenue, Chicago, Ill. 60602.

November 7–10: Association of Clinical Scientists. Shoreham Hotel, Washington. Address: Dr. Robert P. MacFate, Secretary, 125 North Rutledge Street, Pentwater, Mich. 49449.

November 18–21: Southern Medical Association. New Orleans. Address: Mr. Raymond F. Butts, Executive Director, 2601 Highland Avenue, Birmingham, Ala. 35205.

November 21–23: Medical Society of the District of Columbia. Washington Hilton Hotel, Washington. Address: Mr. Thomas Sadler, Executive Director, 2007 Eye Street, N.W., Washington, D. C. 20006.

November 21–26: American Heart Association. Americana Hotel, Bal Harbour, Fla. Address: Mr. Rome A. Betts, Executive Director, 44 East 23rd Street, New York, N. Y. 10010.

December 1–4: American Medical Association (Clinical Convention). Miami Beach, Fla. Address: Dr. Ernest B. Howard, Acting Executive Vice President, 535 North Dearborn Street, Chicago, Ill. 60610.

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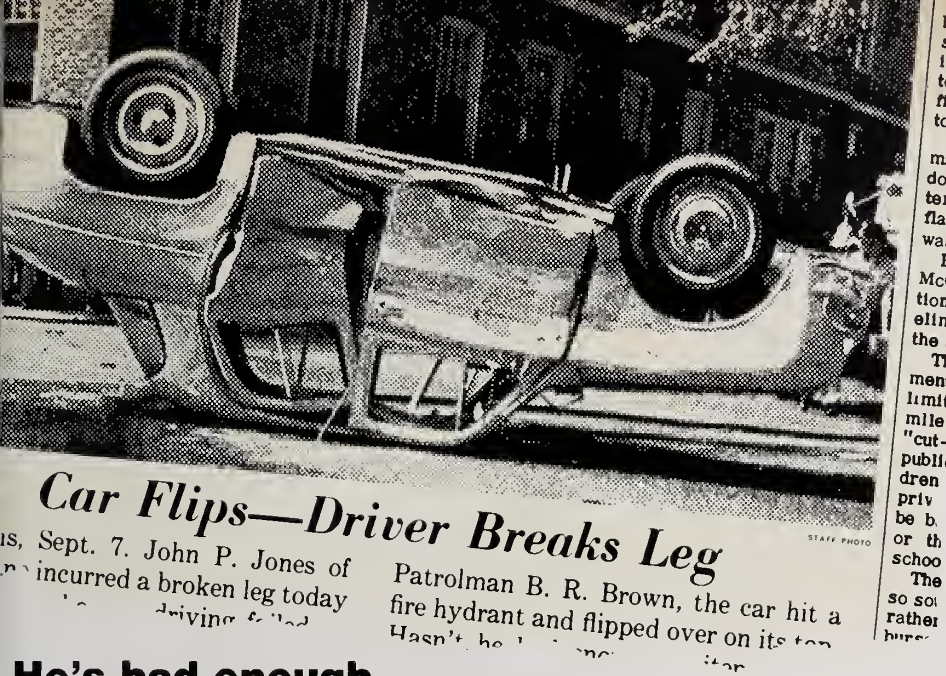

STAFF PHOTO

Car Flips—Driver Breaks Leg

is, Sept. 7. John P. Jones of
incurred a broken leg today

Patrolman B. R. Brown, the car hit a
fire hydrant and flipped over on its

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For the patient who has been through an accident, the worry and anxiety following the experience may actually heighten the perception of pain. This is why there's a classic 1/4 grain *sedative* dose of phenobarbital in Phenaphen with Codeine—*to take the nervous "edge" off,* so the rest of the formula can control the pain more effectively.

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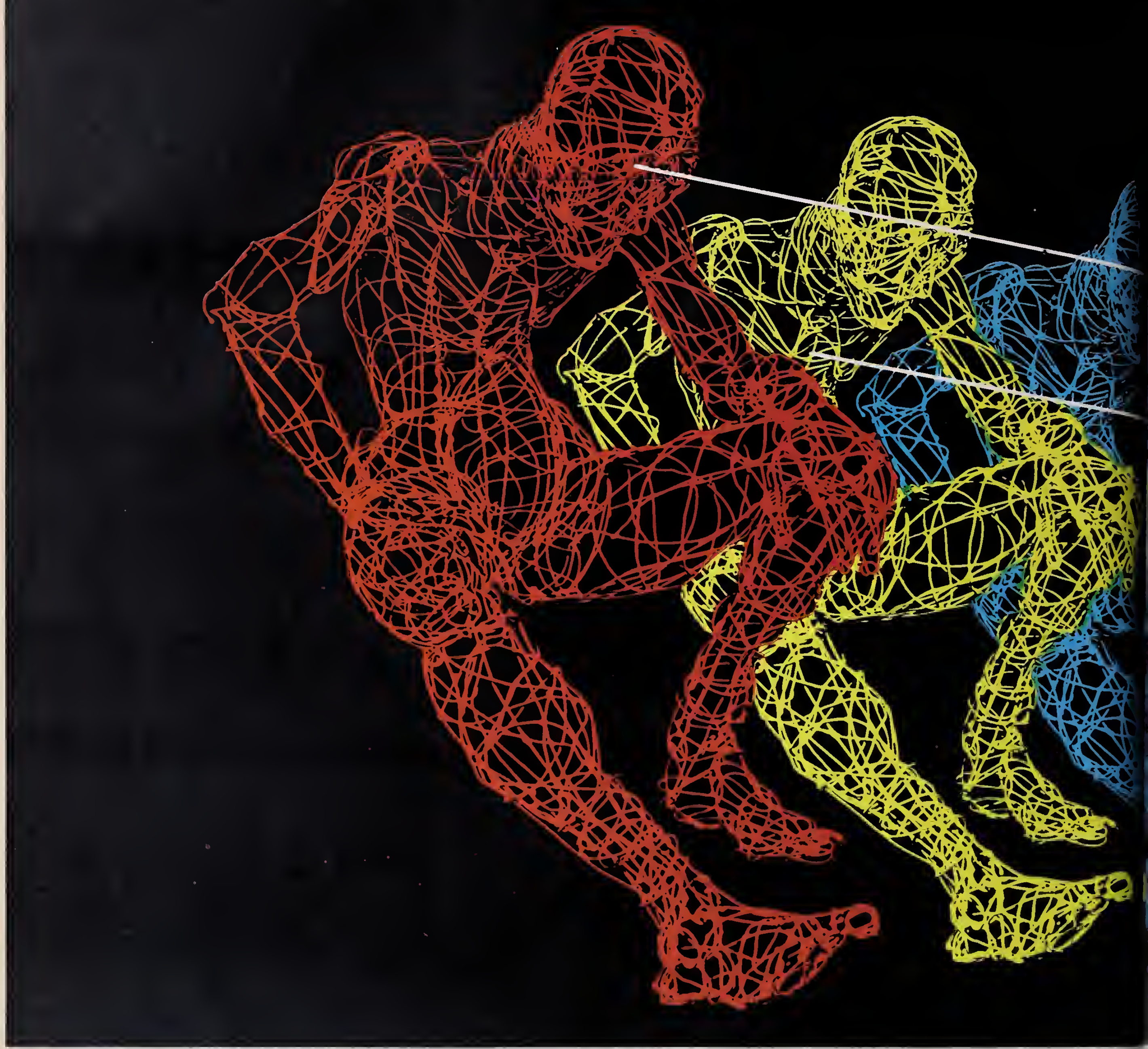

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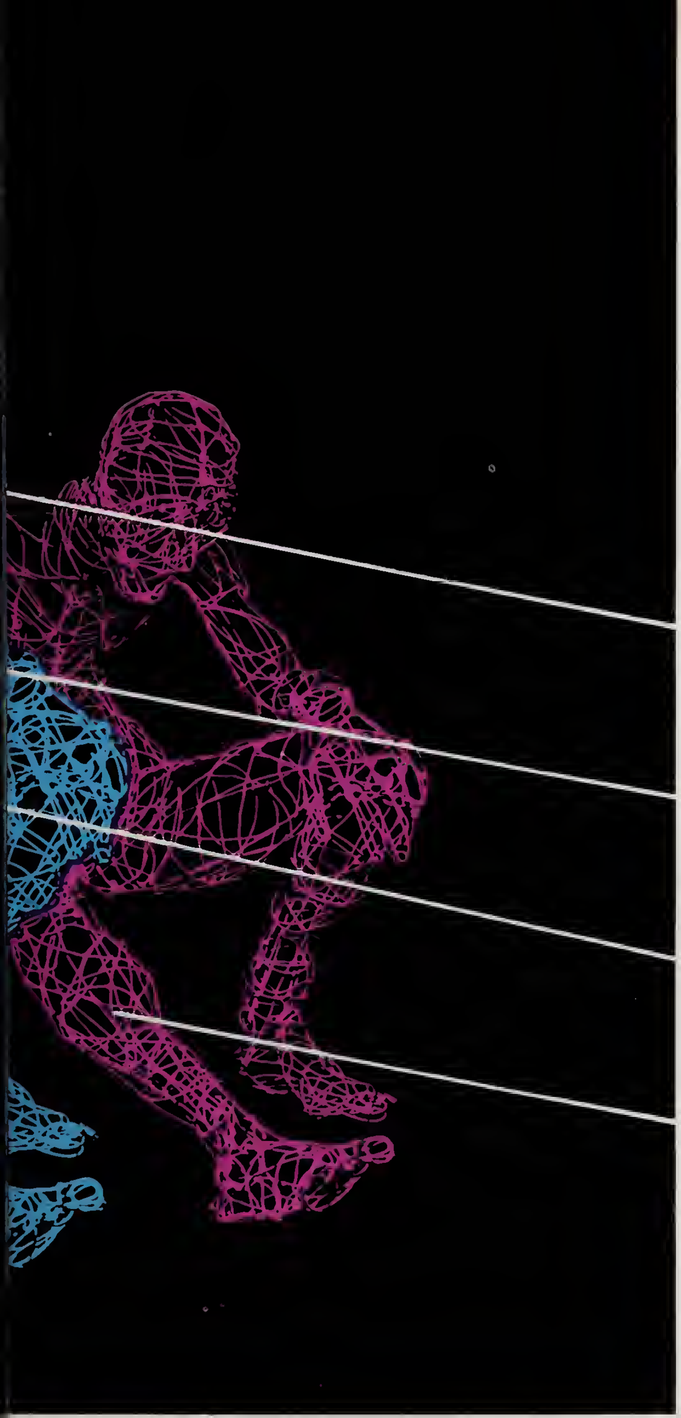

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..because psychic tension may not stop at night

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Precautions: If combined with other psychotropics or anticonvulsants, consider carefully pharmacology of agents employed. Usual precautions indicated in patients severely depressed, or with latent depression, or with suicidal tendencies. Observe usual precautions in impaired renal or hepatic function. Limit dosage to smallest effective amount in elderly and debilitated to preclude ataxia or oversedation.

Side Effects: Drowsiness, confusion, diplopia, hypotension, changes in libido, nausea, fatigue, depression, dysarthria, jaundice, skin rash, ataxia, constipation, headache, incontinence, changes in salivation, slurred speech, tremor, vertigo, urinary retention, blurred vision. Paradoxical reactions such as acute hyperexcited states, anxiety, hallucinations, increased muscle spasticity, insomnia, rage, sleep disturbances, stimulation, have been reported; should these occur, discontinue drug. Isolated reports of neutropenia, jaundice; periodic blood counts and liver function tests advisable during long-term therapy.

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(diazepam)

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VOLUME XXXVII • NUMBER 11

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Leukemia in Pregnancy

V. C. Vitums, M.D., and James G. Sites, M.D.

Idiopathic Hypertrophic Subaortic Stenosis

M. Pooya, M.D., N. Khoi, M.D., R. Sarin, M.D., J. Rios, M.D., and R. A. Massumi, M.D.

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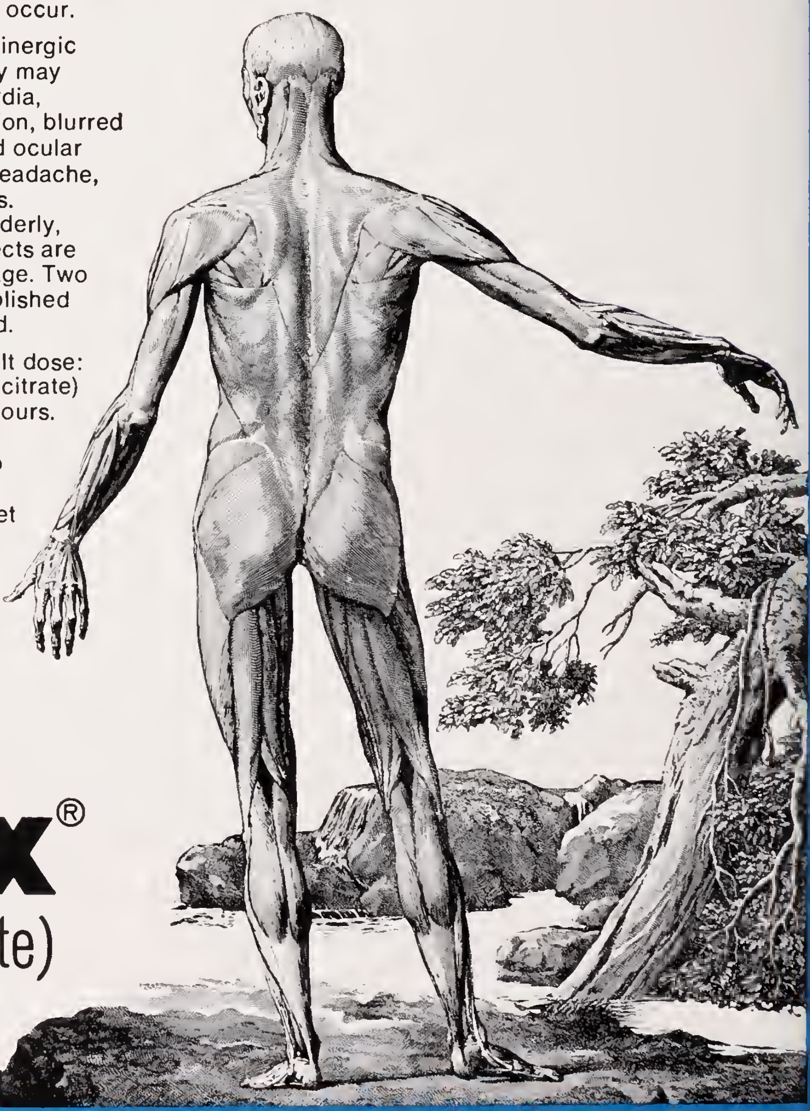
Annual Scientific Assembly

WASHINGTON HILTON HOTEL
November 21–22–23, 1968



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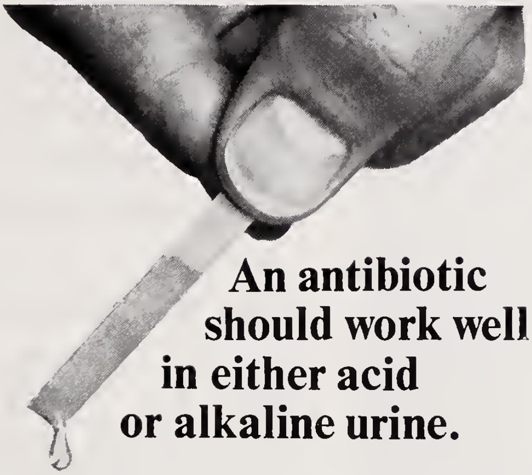

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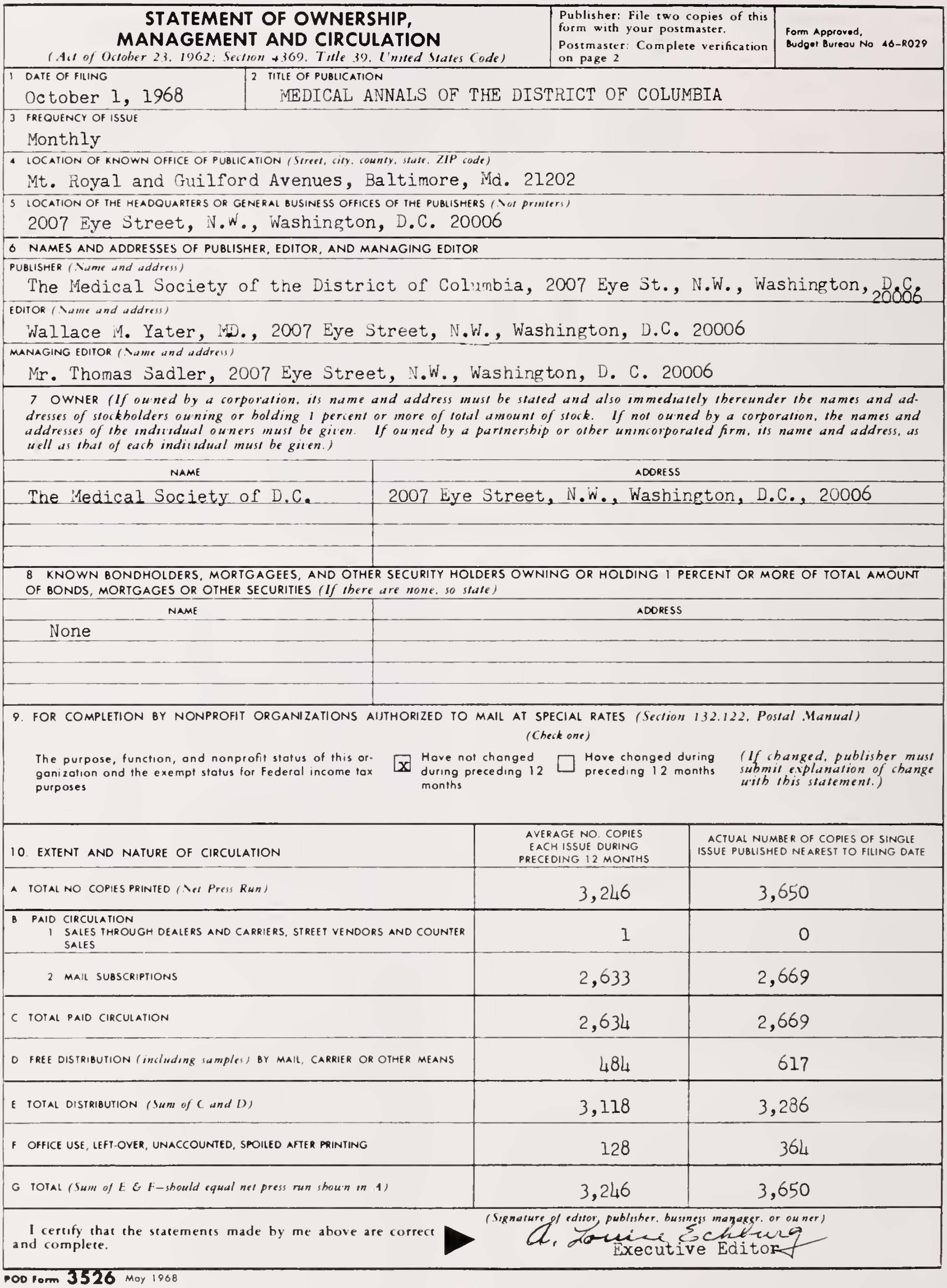

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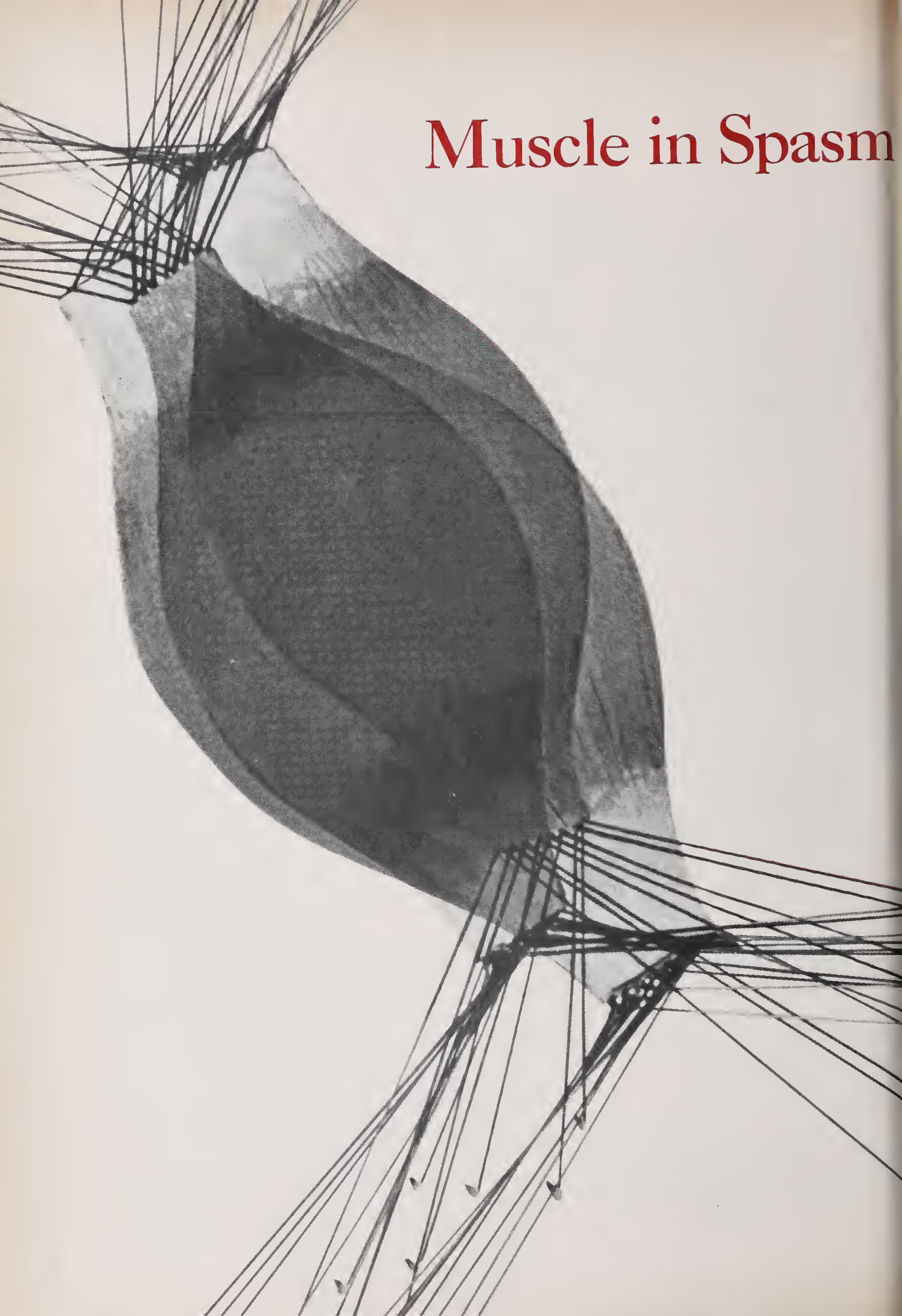

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Contraindicated: Known hypersensitivity to the drug. Children under 6 months of age. Acute narrow angle glaucoma.

Warnings: Not of value in psychotic patients. Caution against hazardous occupations requiring complete mental alertness. When used adjunctively in convulsive disorders, possibility of increase in frequency and/or severity of grand mal seizures may require increased dosage of standard anticonvulsant medication; abrupt withdrawal may be associated with temporary increase in frequency and/or severity of seizures. Advise against simultaneous ingestion of alcohol and other CNS depressants. Withdrawal symptoms have occurred following abrupt discontinuance. Keep addiction-prone individuals under careful surveillance because of their predisposition to habituation and dependence. In pregnancy, lactation or women of childbearing age, weigh potential benefit against possible hazard.

Precautions: If combined with other psychotropics or anticonvulsants, consider carefully pharmacology of agents employed. Usual precautions indicated in patients severely depressed, or with latent depression, or with suicidal tendencies. Observe usual precautions in impaired renal or hepatic function. Limit dosage to smallest effective amount in elderly and debilitated to preclude ataxia or oversedation.

Side Effects: Drowsiness, confusion, diplopia, hypotension, changes in libido, nausea, fatigue, depression, dysarthria, jaundice, skin rash, ataxia, constipation, headache, incontinence, changes in salivation, slurred speech, tremor, vertigo, urinary retention, blurred vision. Paradoxical reactions such as acute hyperexcited states, anxiety, hallucinations, increased muscle spasticity, insomnia, rage, sleep disturbances, stimulation, have been reported; should these occur, discontinue drug. Isolated reports of neutropenia, jaundice; periodic blood counts and liver function tests advisable during long-term therapy.

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Precautions: Antihypertensive therapy with this drug should always be initiated cautiously in postsympathectomy patients and in patients receiving ganglionic blocking agents, other potent antihypertensive drugs or curare. Reduce dosage of concomitant antihypertensive agents by at least one-half. Because of the possibility of progression of renal damage, periodic determination of the BUN is indicated. Discontinue if the BUN rises or liver dysfunction is aggravated. Hepatic coma may be precipitated.

Electrolyte imbalance, sodium and/or potassium depletion may occur. If potassium depletion should occur during therapy, the drug should be discontinued and potassium supplements given, provided the patient does not have marked oliguria.

Take special care in cirrhosis or severe ischemic heart disease and in patients receiving corticosteroids, ACTH, or digitalis. Salt restriction is not recommended.

Adverse Reactions: Nausea, gastric irritation, vomiting, anorexia, constipation and cramping, dizziness, weakness, restlessness, hyperglycemia, glycosuria, hyperuricemia, headache, muscle cramps, orthostatic hypotension, which may be potentiated when chlorthalidone is combined with barbiturates, narcotics or alcohol, aplastic anemia, leukopenia, thrombocytopenia, agranulocytosis, impotence, dysuria, transient myopia, skin rashes, urticaria, purpura, necrotizing angiitis, acute gout, and pancreatitis when epigastric pain or unexplained G.I. symptoms develop after prolonged administration. Other reactions reported with this class of compounds include: jaundice, xanthopsia, paresthesia, and photosensitization.

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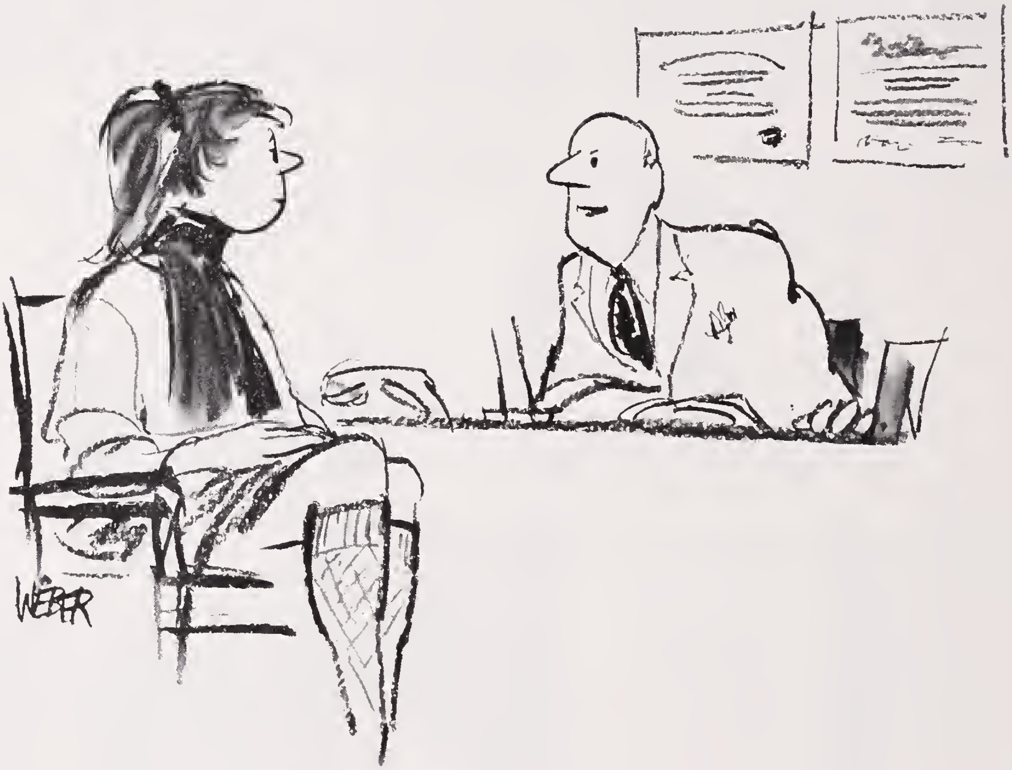

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Dextro-amphetamine sulfate: Excessive use by unstable individuals may result in psychological dependence.

Meprobamate: Careful supervision of dose and amounts prescribed is advised, especially for patients with known propensity for taking excessive quantities of drugs. Excessive and prolonged use in susceptible persons, e.g. alcoholics, former addicts, and other severe psychoneurotics, has been reported to result in dependence on the drug. Where excessive dosage has continued for weeks or months, reduce dosage gradually. Sudden withdrawal may precipitate recurrence of preexisting symptoms such as anxiety, anorexia, or insomnia; or withdrawal reactions such as vomiting, ataxia, tremors, muscle twitching and, rarely, epileptiform seizures. Should meprobamate cause drowsiness or visual disturbances, reduce dosage and avoid operation of motor vehicles, machinery or other activity requiring alertness. Effects of excessive alcohol consumption may be increased by meprobamate. Appropriate caution is recommended with patients prone to excessive drinking. In patients prone to both petit and grand mal epilepsy meprobamate may precipitate grand mal attacks. Prescribe cautiously and in small quantities to patients with suicidal tendencies.

Side Effects: Overstimulation of the central nervous system, jitteriness and insomnia or drowsiness. Dextro-amphetamine sulfate: Insomnia, excitability, and increased motor activity are common and ordinarily mild side effects. Confusion, anxiety, aggressiveness, increased libido, and hallucinations have also been observed, especially in mentally ill patients. Rebound fatigue and depression may follow central stimulation. Other effects may include dry mouth, anorexia, nausea, vomiting, diarrhea, and increased cardiovascular reactivity.

Meprobamate: Drowsiness may occur and can be associated with ataxia; the symptom can usually be controlled by decreasing the dose, or by concomitant administration of central stimulants. Allergic or idiosyncratic reactions: maculopapular rash, acute nonthrombocytopenic purpura with petechiae, ecchymoses, peripheral edema and fever, transient leukopenia. A case of fatal bullous dermatitis, following administration of meprobamate and prednisolone, has been reported. Hypersensitivity has produced fever, fainting spells, angioneurotic edema, bronchial spasms, hypotensive crises (1 fatal case), anuria, stomatitis, proctitis (1 case), anaphylaxis, agranulocytosis and thrombocytopenic purpura, and a fatal instance of aplastic anemia, but only when other drugs known to elicit these conditions were given concomitantly. Fast EEG activity, usually after excessive dosage. Impairment of visual accommodation. Massive overdosage may produce drowsiness, lethargy, stupor, ataxia, coma, shock, vasomotor and respiratory collapse.

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Dextro-Amphetamine Sulfate (15 mg.)
with Meprobamate (300 mg.)

Sustained Release Capsules

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Knee-deep

n osteoarthritic pain

If aspirin doesn't help, move in
ı Tandearil.

The trial period is brief: 1 week.
one tablet q.i.d. at first. Tandearil
ıally starts working within 3 to 4 days.
en response occurs, as little as 1 or
blets daily may hold back pain and
ness, and increase joint motion.

On the next page is a summary
ıdverse reactions, contraindications,
ning and precautions.

Tandearil.
It can help get his mind off his knee.

Please review full Prescribing Information carefully before prescribing.

For osteoarthritic knees, spines, shoulders, hips, etc.:

Tandearil®
oxyphenbutazone

ıdearil, oxyphenbutazone:
brief summary see next page.

Geigy

TA-6006

Tandearil®
oxyphenbutazone

Contraindications: Edema; danger of cardiac decompensation; history or symptoms of peptic ulcer; renal, hepatic or cardiac damage; history of drug allergy; history of blood dyscrasia. The drug should not be given when the patient is senile or when other potent drugs are given concurrently.

Warning: This drug is an analog of phenylbutazone; sensitive patients may be cross-reactive. If coumarin-type anticoagulants are given simultaneously, watch for excessive increase in prothrombin time. Instances of severe bleeding have occurred. Persistent or severe dyspepsia may indicate peptic ulcer; perform upper gastrointestinal x-ray diagnostic tests if drug is continued. Pyrazole compounds may potentiate the pharmacologic action of sulfonylurea, sulfonamide-type agents and insulin. Carefully observe patients receiving such therapy. Use with caution in the first trimester of pregnancy, and in patients with thyroid disease.

Precautions: Before prescribing, carefully select patients, avoiding those responsive to routine measures as well as contraindicated patients. Obtain a detailed history and a complete physical and laboratory examination, including a blood count. The patients should not exceed recommended dosage, should be closely supervised and should be warned to discontinue the drug and report immediately if fever, sore throat, or mouth lesions (symptoms of blood dyscrasia), sudden weight gain (water retention), skin reactions black or tarry stools or other evidence of intestinal hemorrhage occur. Make complete blood counts at weekly intervals during early therapy and at 2-week intervals thereafter. Discontinue the drug immediately and institute countermeasures if the white count changes significantly, granulocytes decrease, or immature forms appear. Use greater care in the elderly and in hypertensives.

Adverse Reactions: The more common are nausea and edema. Swelling of the ankles or face may be minimized by withholding dietary salt, reduction in dosage or use of diuretics. In elderly patients and in those with hypertension, the drug should be discontinued with the appearance of edema. The drug has been associated with peptic ulcer and may reactivate a latent peptic ulcer. The patient should be instructed to take doses immediately after meals or with milk to minimize gastric upset. Drug rash occasionally occurs. If it does, promptly discontinue the drug. Agranulocytosis, exfoliative dermatitis, Stevens-Johnson syndrome, Lyell's syndrome (toxic necrotizing epidermolysis), or a generalized allergic reaction similar to a serum sickness syndrome may occur and require permanent withdrawal of medication. Agranulocytosis can occur suddenly in spite of regular, repeated normal white counts. Stomatitis, salivary gland enlargement, vomiting, vertigo and languor may occur. Leukemia and leukemoid reactions have been reported. While not definitely attributable to the drug, a causal relationship cannot be excluded. Thrombocytopenic purpura and aplastic anemia may occur. Confusional states, agitation, headache, blurred vision, optic neuritis and transient hearing loss have been reported, as have hyperglycemia, hepatitis, jaundice, hypersensitivity angiitis, pericarditis and several cases of anuria and hematuria. With long-term use, reversible thyroid hyperplasia may occur infrequently. Moderate lowering of the red cell count due to hemodilution may occur.

Dosage in Osteoarthritis: Initial: 3 to 6 tablets daily in divided doses. Usually unnecessary to exceed 4 tablets daily. A trial period of one week is considered adequate to determine the therapeutic effect of the drug. Maintenance: Effective level often achieved with 1 or 2 tablets daily, should not exceed 4 tablets daily.

In selecting appropriate dosage in any specific case, consideration should be given to the patient's weight, general health, age and any other factors influencing drug response.

Availability: Tan, round, sugar-coated tablets of 100 mg. in bottles of 100 and 1000.
(B)R-46-800-A

For complete details, please see full Prescribing Information.

Geigy Pharmaceuticals
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Suspected tetracycline-sensitive infection?

While waiting for the results of the sensitivity test, start the therapy likely to succeed...

Although of course it can't replace routine ensitivity testing, your prescription for ACHROMYCIN® V, in a way, provides the ltimate test of therapy under rigorous *in vivo* onditions.

Because ACHROMYCIN® V is effective in reating so many common infections—caused by trains of tetracycline-sensitive organisms—doesn't *stat* dosage of this time-tested antibiotic make good sense?

—Prescribing Information

ACHROMYCIN® V
TETRACYCLINE
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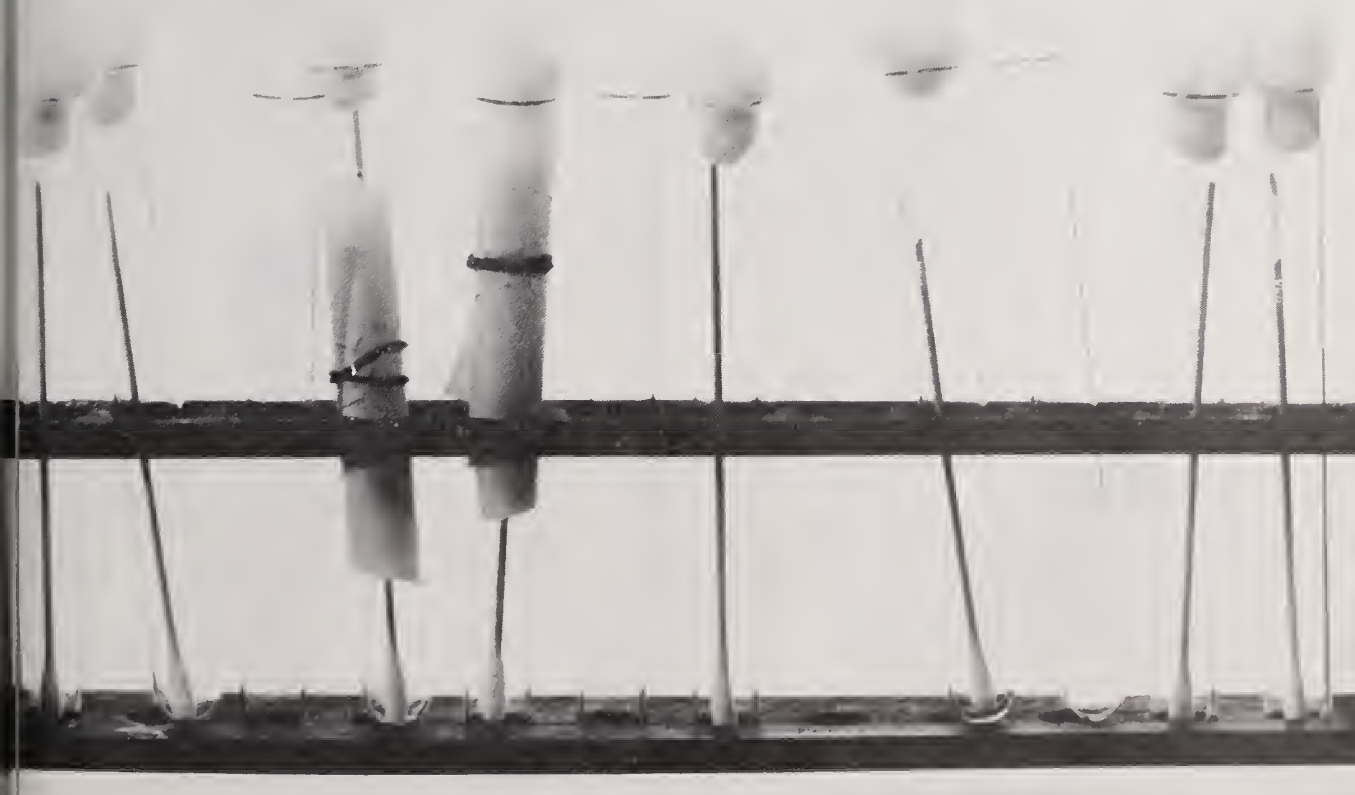
The price differential
is inconsequential.



ACHROMYCIN® V
TETRACYCLINE

Contraindications: Hypersensitivity to tetracycline.

Warning: In renal impairment, since liver toxicity is possible, lower doses are indicated; during prolonged therapy consider serum level determinations. Photodynamic reaction to sunlight may occur in hypersensitive persons. Photosensitive individuals should avoid exposure; discontinue treatment if skin discomfort occurs.

Precautions: Nonsusceptible organisms may overgrow; treat superinfection appropriately. Tetracycline may form a stable calcium complex in bone-forming tissue and may cause dental staining during tooth development (last half of pregnancy, neonatal period, infancy, early childhood).

Side Effects: *Gastrointestinal*—anorexia, nausea, vomiting, diarrhea, stomatitis, glossitis, enterocolitis, pruritus ani. *Skin*—maculopapular and erythematous rashes; exfoliative dermatitis; photosensitivity; onycholysis, nail discoloration. *Kidney*—dose-related rise in BUN. *Hypersensitivity reactions*—urticaria, angioneurotic edema, anaphylaxis. *Intracranial*—bulging fontanels in young infants. *Teeth*—yellow-brown staining; enamel hypoplasia. *Blood*—anemia, thrombocytopenic purpura, neutropenia, eosinophilia. *Liver*—cholestasis at high dosage.

Upon adverse reaction, stop medication and treat appropriately.

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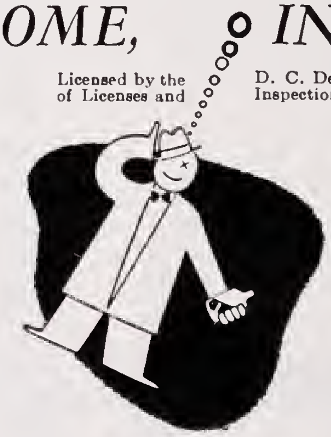

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for the medical management of the acute stages of alcoholism.

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In the complex picture of moderate to severe anxiety...

here is a new reason or prescribing Mellaril (Thioridazine HCl)

ffectiveness in nixed anxiety-depression

ong recognized for its usefulness in the reatment of moderate to severe anxiety, ellaril is now also known to be effective gainst mixed anxiety-depression.

ften the symptoms of anxiety states are ifficult to sort out—even with the most careful robing. The patient may manifest symptoms of gitation, restlessness, insomnia, somatic omplaints. But what of the depression that may e mixed in the total picture? It is reassuring o know that Mellaril may be prescribed—with trong possibilities of success—when there is nxiety alone or a mixture of anxiety nd depression.

Before prescribing or administering, see Sandoz literature for full product information, including adverse reactions reported with phenothiazines. The following is a brief precautionary statement.

Contraindications: Severe central nervous system depression, comatose states from any cause, hypertensive or hypotensive heart disease of extreme degree.

Warnings: Administer cautiously to patients who have previously exhibited a hypersensitivity reaction (e.g., blood dyscrasias, jaundice) to phenothiazines. Phenothiazines are capable of potentiating central nervous system depressants (e.g., anesthetics, opiates, alcohol, etc.) as well as atropine and phosphorus insecticides. During pregnancy, administer only when necessary.

Precautions: There have been infrequent reports of leukopenia and/or agranulocytosis and convulsive seizures. In epileptic patients, anticonvulsant medication should also be maintained. Pigmentary retinopathy may be avoided by remaining within the recommended limits of dosage. Administer cautiously to patients participating in activities requiring complete mental alertness (e.g., driving). Orthostatic hypotension is more common in females than in males. Do not use epinephrine in treating drug-induced hypotension. Daily doses in excess of 300 mg. should be used only in severe neuropsychiatric conditions.

Adverse Reactions: *Central Nervous System*—Drowsiness, especially with large doses, early in treatment; infrequently, pseudoparkinsonism and other extrapyramidal symptoms; nocturnal confusion, hyperactivity, lethargy, psychotic reactions, restlessness, and headache. *Autonomic Nervous System*—Dryness of mouth, blurred vision, constipation, nausea, vomiting, diarrhea, nasal stuffiness, and pallor. *Endocrine System*—Galactorrhea, breast engorgement, amenorrhea, inhibition of ejaculation, and peripheral edema. *Skin*—Dermatitis and skin eruptions of the urticarial type, photosensitivity. *Cardiovascular System*—Changes in the terminal portion of the electrocardiogram have been observed in some patients receiving the phenothiazine tranquilizers, including Mellaril (thioridazine hydrochloride). While there is no evidence at present that these changes are in any way precursors of any significant disturbance of cardiac rhythm, several sudden and unexpected deaths apparently due to cardiac arrest have occurred in patients previously showing electrocardiographic changes. The use of periodic electrocardiograms has been proposed but would appear to be of questionable value as a predictive device. *Other*—A single case described as parotid swelling.

Mellaril®
(Thioridazine HCl)
25 mg. t.i.d.

for moderate to severe anxiety and mixed anxiety-depression

SANDOZ SANDOZ PHARMACEUTICALS, HANOVER, N. J. 68-170

Louie!
You're
back.

Louie lost weeks with acute shoulder bursitis. That's a l
of pain, stiffness and tenderness...and also a lot of fis
It might have been different with **Butazolidin® alk**

100 mg. phenylbutazone
100 mg. dried aluminum hydroxide gel
150 mg. magnesium trisilicate

If it doesn't work in a week, forget

But please don't forget this:

Contraindications: Edema; danger of cardiac decompensation; history or symptoms of peptic ulcer; renal, hepatic or cardiac damage; history of drug allergy; history of blood dyscrasia. The drug should not be given when the patient is senile or when other potent drugs are given concurrently. Large doses of the alka formulation are contraindicated in glaucoma.

Warning: If coumarin-type anticoagulants are given simultaneously, watch for excessive increase in prothrombin time. Instances of severe bleeding have occurred. Persistent or severe dyspepsia may indicate peptic ulcer; perform upper gastrointestinal x-ray diagnostic tests if drug is continued. Pyrazole compounds may potentiate the pharmacologic action of sulfonylurea, sulfonamide-type agents and insulin. Carefully observe patients receiving such therapy. Use with caution in the first trimester of pregnancy and in patients with thyroid disease.

Precautions: Before prescribing, carefully select patients, avoiding those responsive to routine measures as well as contraindicated patients. Obtain a detailed history and a complete physical and laboratory examination, including a blood count. Patients should not exceed recommended dosage, should be closely supervised and should be warned to discontinue the drug and report immediately if fever, sore throat, or mouth lesions (symptoms of blood dyscrasia); sudden weight gain (water retention); skin reactions; black or tarry stools or other evidence of intestinal hemorrhage occur. Make complete blood counts at weekly intervals during early therapy and at 2-week intervals thereafter. Discontinue the drug immediately and institute countermeasures if the white count changes significantly, granulocytes decrease, or immature forms appear. Use greater care in the elderly and in hypertensives.

Adverse Reactions: The more common are nausea and edema. Swelling of the ankles or face may be minimized by withholding dietary salt, reduction in dosage or use of diuretics. In elderly patients and in those with hypertension the drug should be discontinued with the appearance of edema. The drug has been associated with peptic ulcer and may reactivate a latent peptic ulcer. The patient should be instructed to take doses immediately before or after meals or with milk to minimize gastric upset. Drug rash occasionally occurs. If it does, promptly discontinue the drug. Agranulocytosis, exfoliative dermatitis, Stevens-Johnson syndrome, Lyell's syndrome (toxic necrotizing epidermolysis), or a generalized allergic reaction similar to serum sickness may occur and require permanent withdrawal of medication. Agranulocytosis can occur suddenly in spite of regular, repeated normal white counts. Stomatitis, salivary gland enlargement, vomiting, vertigo and languor may occur. Leukemia and leukemoid reactions have been reported. While not definitely attributable to the drug, a causal relationship cannot be excluded. Thrombocytopenic purpura and aplastic anemia may occur. Confusional states, agitation, headache, blurred vision, optic neuritis and transient hearing loss have been reported, as have hyperglycemia, hepatitis, jaundice, hypersensitivity angiitis, pericarditis and several cases of anuria and hematuria. With long-term use, reversible thyroid hyperplasia may occur infrequently. Moderate lowering of the red cell count due to hemodilution may occur.

Dosage in Painful Shoulder: Initial: 3 to 6 capsules daily in 3 or 4 equal doses. Trial period: 1 week. Maintenance dosage should not exceed 4 capsules daily; response is often achieved with 1 or 2 capsules daily.

In selecting the appropriate dosage in any specific case, consideration should be given to the patient's weight, general health, age and any other factors influencing drug response.

(B) 46-070-B

**For complete details,
please see full prescribing information.**

Geigy Pharmaceuticals
Division of Geigy Chemical Corporation
Ardsley, New York 10502

Butazolidin® alka Geigy

Capsules
100 mg. phenylbutazone
100 mg. dried aluminum hydroxide gel
150 mg. magnesium trisilicate

BU-6210

The Divorced Depressive: "Forty, Forgotten

"Had a large wedding."
"Had a fine home and family."

.Forlorn”

d a divorce.”
a real loser,
d so are
kids.”

The loss of marital compatibility results in nearly 500,000 U.S. divorces each year.

Depression characterized by untold guilt feelings, grief and loneliness may follow.[1]

When you diagnose depression, Tofrānil may be indicated for relief.

As maintenance therapy during the active phase of depression, Tofrānil can often help prevent relapse.

The use of Tofrānil in patients receiving M.A.O.I.'s is contraindicated. In patients with cardiovascular disease, thyroid disorders, increased intraocular pressure; in those receiving anticholinergics (including antiparkinsonism agents), thyroid medication or antihypertensive adrenergic neuron-blocking agents; and in those in their first trimester of pregnancy—the special precautions listed in the Prescribing Information should be carefully observed.

Toxic reactions severe enough to require discontinuation of Tofrānil are uncommon. However, for complete details, please refer to the complete Prescribing Information.

Turn page for brief summary of Prescribing Information.

in depression

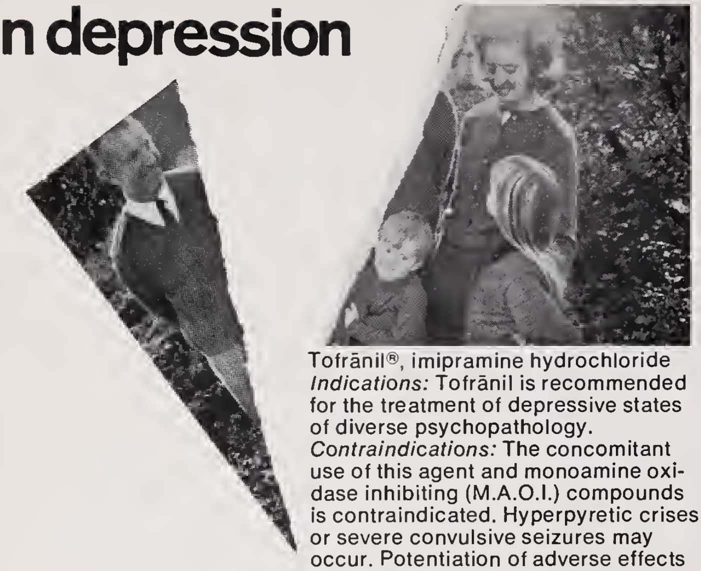
Tofrānil® imipramine hydrochloride

Geigy

in depression

Tofrānil® Geigy

imipramine
hydrochloride



Tofrānil®, imipramine hydrochloride
Indications: Tofrānil is recommended for the treatment of depressive states of diverse psychopathology.
Contraindications: The concomitant use of this agent and monoamine oxidase inhibiting (M.A.O.I.) compounds is contraindicated. Hyperpyretic crises or severe convulsive seizures may occur. Potentiation of adverse effects can be serious or even fatal. An interval of at least 7 days after M.A.O.I. therapy has been discontinued should be allowed before this drug may be substituted. Initial dosage should be low, increases should be gradual, and the patient's progress should be carefully observed.
Warning: Clinical reports have suggested that there may be a risk of teratogenesis associated with the use of this compound during the first trimester of pregnancy. Unless, in the opinion of the prescribing physician, the potential benefits outweigh the possible risks, it should not be used during the first trimester of pregnancy.
Cardiovascular complications, including myocardial infarction and arrhythmias, have occasionally occurred in susceptible individuals. Patients with cardiovascular disease should be given the drug only under careful observation and in low dosage.
Precautions: Since suicide is always a possibility in severely depressed patients and one which may persist until significant remission occurs, such patients should be carefully supervised during early treatment. Some severely depressed patients may also require hospitalization and/or concomitant electroconvulsive therapy.
Because of its anticholinergic effect, caution should be observed in prescribing the drug for patients with increased intraocular pressure.
In rare instances, transient cardiac arrhythmias have occurred in hyperthyroid patients and in patients receiving thyroid medication when this compound was added to the regimen. Imipramine may block the pharmacologic activity of guanethidine and other related adrenergic neuron-blocking agents.
The drug is not recommended at the present time in patients under 12 years of age.
Adverse Reactions: Dryness of the mouth, tachycardia, constipation, disturbances of accommodation, sweating, dizziness, weight gain, urinary frequency or retention, nausea and vomiting, peripheral neuritis, mild parkinson-like syndrome, tremors, rare cases of falling in elderly patients, confusional states (with such symptoms as hallucinations and disorientation), activation of psychosis in schizophrenics and agitation (including hypomanic and manic episodes) which may require dosage reduction and/or addition of a tranquilizer or temporary discontinuation of the drug, epileptiform seizures, orthostatic hypotension and substantial blood pressure fall in hypertensive patients, purpura, transient jaundice, bone marrow depression including agranulocytosis, sensitization and skin rash including photosensitization, eosinophilia, and mild withdrawal symptoms on sudden discontinuation after prolonged treatment with high doses.
Occasional hormonal effects (impotence, decreased libido, and estrogenic effects) may be observed.
Atropine-like effects may be more pronounced (e.g. paralytic ileus) in susceptible patients and in those using anticholinergic agents (including antiparkinsonism drugs).
Outpatient Adult Dosage: Initially, 75 mg. daily, increased, if necessary, to 150 or 200 mg. Maintenance dosage may be lower, 50 to 150 mg. daily, if possible.
Geriatric and Adolescent Dosage: Initially, 30 or 40 mg. daily, which may be increased according to response and tolerance. It is usually unnecessary to exceed 100 mg. daily.
A lag in therapeutic response, lasting from a few days to a few weeks, should be expected. When dosage recommendations are already being followed, increasing the dosage does not normally shorten this latency period and may increase the incidence of adverse reactions.
Availability: Round tablets of 25 and 50 mg.; triangular tablets of 10 mg. for geriatric and adolescent use; and ampuls, each containing 25 mg. in 2 cc. for I.M. administration.
(B)R-46-850-C

For complete details, please refer to the full Prescribing Information.

Geigy Pharmaceuticals
Division of Geigy Chemical Corporation
Ardsley, New York 10502

TO-6757

PAMPHLETS

AMA Publications

(Available from the Order Department, American Medical Association, 535 North Dearborn Street, Chicago, Ill. 60610. Single copies of most of these are available free to physicians upon request.)

Amphetamines. 4 pp. **Barbiturates.** 4 pp. **Glue Sniffing.** 2 pp. **LSD.** 2 pp. **Marihuana.** 2 pp. They describe effects of these dangerous substances on the body; stress hazards of experimentation, psychological effects on users, and legal controls. Price, 15 cents each.

Dandruff. Reprinted from *Today's Health* 1968. Developed to enlighten the public concerning dandruff, its suspected causes and possible control. "Brand-name Guide to Antidandruff Products" included in pamphlet. 8 pp. Price, 20 cents.

The Illness Called Alcoholism. Written for the laity. 14 pp. Price, 20 cents.

Johnny Makes the Team. Information on health aspects of athletic programs. 6 pp. Price, 10 cents.

The Manual on Alcoholism. Physician guide outlining major factors in causes, diagnosis, and treatment of alcoholism and medical management of alcoholic patients. 100 pp. Price, 50 cents.

Your Body and How It Works. Written in language of a child so that parent and child may read it together. Fosters wholesome attitude toward the body and encourages interest in preserving it. 30 pp. Price, 45 cents.

Miscellaneous

Bibliography on Biopharmaceutics. Contains abstracts and journal references on influence of pharmaceutical formulation on therapeutic activity of drugs. 64 pp. Pharmaceutical Manufacturers Association, 1155 15th Street, N.W., Washington, D. C. 20005.

An Environment Fit for People (P.A.P. No. 421). Raymond F. Dasmann. Outlines problems of the cities in terms of living space, transportation, provision of water, outdoor recreation, and especially pollution. 28 pp. Price, 25 cents. Public Affairs Committee, 381 Park Avenue South, New York, N.Y. 10016.

Medicaid: The People's Health Plan (P.A.P. No. 422). Raymond S. Alexander and Simon Podair. Outlines progress made to meet lack of medical care for Nation's poor through Federal funds "for a medical assistance program." 20 pp. Price, 25 cents. Public Affairs Committee, 381 Park Avenue South, New York, N. Y. 10016.

One by one
the family's downed
Because the
G.I. bug's around

Parepectolin for quick relief of acute diarrhea
...soothes colicky pain with paregoric*
...consolidates fluid stools with pectin
...adsorbs irritants with kaolin,
and protects intestinal mucosa.

Whether it's a 24-hour "bug", a food problem, or simply nervousness and anxiety, Parepectolin will bring the diarrhea under control until etiology can be determined. In some cases, Parepectolin may be all the therapy necessary.

Parepectolin®

Each fluid ounce of creamy white suspension contains:
*Paregoric (equivalent) .... (1.0 dram) 3.7 ml.
Contains opium (¼ grain) 15 mg. per fluid ounce.
warning: may be habit forming
Pectin .... (2½ grains) 162 mg.
Kaolin (specially purified) .... (85 grains) 5.5 Gm.
(alcohol 0.69%)
Usual Adult Dose: One or two tablespoonfuls three times daily.
Usual Children's Dose: One or two teaspoonfuls three times daily.

RORER
WILLIAM H. RORER, INC.
Fort Washington, Pa.

It's almost as if you were there to give an injection of penicillin

V-Cillin K®, Pediatric dependable oral penicillin therapy

Potassium Phenoxymethyl Penicillin

Description: V-Cillin K, the potassium salt of V-Cillin® (phenoxymethyl penicillin, Lilly), combines acid stability with immediate solubility and rapid absorption. Higher, more rapid serum levels are obtained than with equal oral doses of penicillin G.

Indications: Streptococcus, pneumococcus, and gonococcus infections; infections caused by sensitive strains of staphylococci; prophylaxis of streptococcus infections in patients with a history of rheumatic fever; and prevention of bacterial endocarditis after tonsillectomy and tooth extraction in patients with a history of rheumatic fever or congenital heart disease.

Contraindication: Penicillin hypersensitivity.

Warnings: In rare instances, penicillin may cause acute anaphylaxis which may prove fatal unless promptly controlled. This type of reaction appears more frequently in patients with a history of sensitivity reactions to penicillin or with bronchial asthma or other allergies. Resuscitative drugs should be readily available. These include epinephrine and pressor drugs (as well as oxygen for inhalation) for immediate allergic manifestations and antihistamines and corticosteroids for delayed effects.

Precautions: Use cautiously, if at all, in a patient with a strongly positive history of allergy.

In prolonged therapy with penicillin, and particularly with high parenteral dosage schedules, frequent evaluation of the renal and hematopoietic systems is recommended.

In suspected staphylococcus infections, proper laboratory studies (including sensitivity tests) should be performed.

The use of penicillin may be associated with the overgrowth of penicillin-insensitive organisms. In such cases, discontinue administration and take appropriate measures.

Adverse Reactions: Although serious allergic reactions are much less common with oral penicillin than with intramuscular forms, manifestations of penicillin allergy may occur.

Penicillin is a substance of low toxicity, but it possesses a significant index of sensitization. The following hypersensitivity reactions have been reported: skin rashes ranging from maculopapular eruptions to exfoliative dermatitis; urticaria; and reactions resembling serum sickness, including chills, fever, edema, arthralgia, and prostration. Severe and often fatal anaphylaxis has occurred (see Warnings). Hemolytic anemia, leukopenia, thrombocytopenia, and nephropathy are rarely observed side-effects and are usually associated with high parenteral dosage.

Administration and Dosage: Usual dosage range, 125 mg. (200,000 units) three times a day to 500 mg. (800,000 units) every four hours. For infants, 50 mg. per Kg. per day divided into three doses.

See package literature for detailed dosage instructions for prophylaxis of streptococcus infections, surgery, gonorrhea, and severe infections.

How Supplied: Tablets V-Cillin K® (Potassium Phenoxymethyl Penicillin Tablets, U.S.P.), 125 mg. (200,000 units), 250 mg. (400,000 units), and 500 mg. (800,000 units).

V-Cillin K® (potassium phenoxymethyl penicillin, Lilly), Pediatric, for Oral Solution, 125 mg. (200,000 units) and 250 mg. (400,000 units) per 5 cc. of solution (approximately one teaspoonful). [042567A]

800198

Additional information available to physicians upon request.

Eli Lilly and Company, Indianapolis, Indiana 46206

Lilly

MEDICAL ANNALS
of the District of Columbia

November 1968
VOLUME XXXVII • NUMBER 11

The George Washington University Number

THIS IS THE SEVENTH of a fifth series of special numbers of the MEDICAL ANNALS, the scientific material for which is supplied by medical institutions and scientific organizations in the Washington Metropolitan Area. For the GEORGE WASHINGTON UNIVERSITY NUMBER the Editorial Board is indebted to Dr. Alvin E. Parrish, Consulting Editor of the MEDICAL ANNALS, who secured the papers which appear in this issue.

Factors Influencing the Trend in Antimicrobial Therapy

PAUL G. COHEN, M.D.,* AND MONROE J. ROMANSKY, M.D., F.A.C.P.†

THE IMPACT produced by the increasing number of newer and more potent antimicrobial agents upon the management and control of many infectious diseases has been dramatic. While this has given the physician a wider choice, allowing him to tailor his antimicrobial therapy more effectively to specific diseases, it has increased the need for more definitive microbiology and specific diagnosis. In addition, the problem of untoward reactions and the emergence of resistant bacteria underscore the responsibility of the physician for discriminate use of these agents. Effective antimicrobial therapy is dependent upon a variety of considerations which influence the ultimate prognosis but basically is a complicated relationship between the host, the invader, and the antimicrobial agent. In the consideration of these areas some basic principles can be established which should lead to a rational approach to the use of these antimicrobial agents.

Mechanisms of Action of Antimicrobial Agents

In recent years a great deal of knowledge has been assembled through the use of antimicrobial agents as biological probes. At present, certain of the antimicrobial agents are thought to exert their effects at particular loci. In general, antimicrobial agents can be said to inhibit cell-wall synthesis, inhibit protein synthesis, or alter the permeability of the cell membrane. Several of the more toxic agents like actinomycin inhibit RNA synthesis or, like gramicidin, uncouple oxidative phosphorylation. These aspects have been extensively reviewed and can be consulted for greater detail elsewhere, since they are beyond the scope of the present general discussion.[1-5]

Many of the antibiotics which have a "bactericidal" action seem to affect either the cell wall or the cell membrane. The synthesis of the bacterial cell wall is a complex phenomenon and is subject to the inhibition at critical loci by different antibiotics. Cycloserine, which functions like a structural analogue of D-alanine, interrupts the formation of an important pentapeptide by inhibiting the incorporation of this amino acid. Further along the sequence of cell-wall synthesis the glycopeptide antibiotics such as ristocetin and vancomycin probably block the transfer of the preformed glycopeptide subunits through the cell membrane. In the final stage of cell-wall synthesis the penicillins and the cephalosporins prevent the cross linking of adjacent glycopeptide chains.

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* Formerly Chief Resident, the George Washington University Medical Division, District of Columbia General Hospital; present address: Treponematosis Section, Venereal Disease Research Laboratory, National Communicable Disease Center, Atlanta, Ga. 30333.

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Another group of antibiotics function by altering the function of the cell membrane. Included here are several different types of antibiotics. The polyene antifungal antibiotics (amphotericin B and nystatin) combine with the sterols in the cell membranes (and also in the cell membranes of human erythrocytes). The polymyxins (polymyxin B and colistin or polymyxin E) are a group of cationic detergents which disrupt the osmotic integrity of the cell, presumably by attaching to various anionic binding sites.

In recent years research into the genetic control of protein synthesis has resulted in a greater understanding of the actions of certain antibiotics which are now known to exert their influence on protein synthesis. Several of these important antibiotics appear to operate at specific ribosomal loci. The aminoglycoside antibiotics (streptomycin, neomycin, and kanamycin) are thought to attach to the 30 S ribosomal subunit and seemingly result in misreading of the genetic code. On the other hand, the tetracyclines, chloramphenicol, erythromycin, and lincomycin also effect the translation of genetic information but result in protein-synthesis inhibition rather than in the production of defective or "nonsense protein." Curiously, the latter group of antibiotics generally exert a "bacteriostatic" effect, whereas the aminoglycoside group generally are considered to be "bactericidal."

In addition to the molecular mechanisms of action, several basic principles of antimicrobial therapy should be noted. Antimicrobial agents require a definite interval of exposure for effective action. Furthermore, they act chiefly on multiplying microörganisms. During this period of time effective drug concentrations are reached, and sufficient contact with the microörganism at the optimum point in its metabolic cycle is required to produce the desired effects.

The time interval between the clinical response and the initiation of therapy will obviously vary. A patient will have a rapid or a delayed response depending upon the size of the inoculum and the anatomic or mechanical factors as well as the patient's total immune response and the dose of antibiotic employed.

These factors have practical importance in clinical medicine, particularly in deep-seated infections, in which the microörganism, although sensitive, may lie in areas where it is difficult to achieve significant tissue concentrations of antibiotics. This is particularly true in cases of subacute bacterial endocarditis and osteomyelitis, where a bactericidal effect is essential. The rationale for a bactericidal agent lies in the fact that with such agents (penicillin, cephalothin, streptomycin, polymyxins) the inhibitory concentration and the lethal concentrations are rather close. In the case of those antibiotics generally considered bacteriostatic (chloramphenicol, the tetracyclines, and erythromycin) the gap between the inhibitory concentration and the lethal concentration is greater, and more adequate host defenses are necessary.

Changing Patterns of Bacterial Resistance

The emergence of antibiotic-resistant bacteria remains one of the problems in the field of antimicrobial therapy.[6] This problem becomes more pronounced when one deals with chronic infections such as endocarditis, pyelonephritis, and osteomyelitis. Often it is the presence of a foreign body or the failure to achieve adequate drainage in the area of suppuration that leads to the apparent therapeutic failure.

Of profound significance is the spector of infectious drug resistance now known to be carried by an extrachromosomal genetic resistance factor (R factor).[7, 8] Until recently the predominant mechanism of antibiotic resistance was thought to be the occurrence of spontaneous mutation and subsequent selection of resistant strains during therapy. It has been conclusively demonstrated that multiple drug resistance can be directly transferred from resistant enteric bacteria to sensitive strains. Certain R factors (extrachromosomal genetic DNA) are responsible for this infectious drug resistance. The R factor contains a "resistance-transfer factor" (RTF) which determines the transfer of drug resistance. A similar type of circumstance seems to occur with multiple antibiotic-resistant staphylococci. It differs, however, in that staphylococcal DNA cannot be transferred by direct cell-to-cell contact. In this instance, resistance is carried by plasmids (cytoplasmic genes) and transferred by certain bacteriophages.[9]

The widespread use of antimicrobial agents coupled with increase in the number of resistant microörganisms suggests the disturbing possibility that some of the currently available antibiotics may become less effective. It should be noted that the routine blanket use of antibiotics in livestock feeding not only exposes us to these agents but also may serve to facilitate the production of antibiotic-resistant strains.

TABLE 1: Pattern of Bacterial Sensitivity to the Antibiotics Tendency toward Resistance or Development of Resistance

Rarely	Occasionally	Frequently
Group A beta-hemolytic streptococcus	Alpha-hemolytic streptococcus	Micrococcus (staphylococcus)
Pneumococcus*	Nonhemolytic streptococcus	Enterococcal group
Meningococcus†		Proteus (indol negative and positive)
Gonococcus‡		*Pseudomonas aeruginosa*
H. influenzae		Coli-aerogenes group
Treponemes		*Mycobacterium tuberculosis*

* Occasional resistance to tetracyclines, erythromycin, lincomycin,

† Resistance developing to sulfonamides (group B and C).

‡ Resistant to sulfonamides; relative resistance to penicillin.

Although certain microörganisms have a propensity to develop antibiotic resistance, others have shown little evidence of this (table 1). The group A beta hemolytic streptococcus, pneumococcus, meningococcus, gonococcus, treponemes and *Hemophilus influenzae* will respond readily when adequate amounts of a single antibiotic have been used for a sufficient period of time. Instances of the pneumococcus developing resistance to tetracycline,[10, 11] erythromycin,[12, 13] and lincomycin[12, 13] have been noted. Recent reports also indicate that many isolates of the meningococcus, particularly sero-groups B and C, have become resistant to the sulfonamides.[14, 15] The relative resistance of some strains of the gonococcus to penicillin G has also been observed.[16]

As also noted in table 1, the microörganisms that have most frequently developed antibiotic resistance in clinical medicine have been the staphylococci, members of the gram-negative group, strains of *Mycobacterium tuberculosis*, and the enterococcal group.

Staphylococcal resistance to penicillin G is most frequently associated with penicillinase-producing organisms, but certain universal strains may be inherently resistant. Generally, the penicillinase-resistant semisynthetic penicillins, as well as the newer agents cephalothin and lincomycin, are effective.

Combinations of Antibiotics

The interaction of antibiotics in combination, particularly as they relate to the microörganism and the host, has not been well delineated. The magnitude of multiple antibiotic therapy has been indicated in a study by Cluff and colleagues[17] who noted that 11 per cent of the patients hospitalized at the Johns Hopkins Hospital received from 2 to 5 antibiotics, whereas 18 per cent received a single antibiotic.

In a recent review Dowling[18] pointed out that combination therapy is frequently employed without the necessary regard for the interaction of these agents. Despite these negative aspects, certain combinations of antibiotics are extremely useful when employed for the proper indications.

Combinations of penicillin and streptomycin were first demonstrated to be synergistic in vitro[19] and were subsequently employed in the treatment of enterococcal endocarditis.[20] Subsequently these combinations were employed successfully in a number of infections and particularly in the short-term therapy of alpha-hemolytic streptococcal endocarditis.[21]

Antibiotic antagonism has been suggested, particularly in pneumococcal meningitis in which bacteriostatic or suppressive agents such as the tetracyclines or chloramphenicol have been used in conjunction with a bactericidal agent such as penicillin.[22, 23] It is likely that microbial suppression by the bacteriostatic agent minimizes the multiplication rate of the microörganisms with a resultant decrease in effectiveness of the bactericidal agent. This emphasizes the necessity for using a single antibiotic in an adequate dose for an adequate period of time in those disease entities in which a sensitive microörganism is present.

In some clinical situations combinations of antimicrobial agents may be indicated. As noted in table 2, those microörganisms which frequently tend to develop resistance may be effectively controlled by a combination of antibiotics because of their different mode of action, but the newer broad-spectrum agents, cephalothin, cephaloridine and ampicillin, may lessen the need for combination therapy since these may be used effectively singly.

Although streptomycin is rarely used alone, it appears to be the common denominator of combination therapy. Tuberculosis is a prime example for which the use of combined therapy is mandatory and suppresses the development of resistance. With single therapy the tubercle bacillus develops resistance with relative rapidity.[24] Acute brucellosis is another example in which streptomycin and one of the tetracycline group are more effective than either alone.

TABLE 2: Infections Which May Require 2 Antimicrobial Agents

Tuberculosis	Streptomycin and PAS or isoniazid; or PAS and isoniazid
Brucellosis (acute)	Streptomycin and one of the tetracycline group
Bacterial endocarditis:	
A-hemolytic streptococcus	Penicillin and streptomycin
Enterococcal	Penicillin and streptomycin; ampicillin alone may be effective
Pneumonia (Klebsiella or Friedländer's)	Streptomycin and one of the tetracycline group or chloramphenicol; cephalothin or cephaloridine alone may be effective
Pseudomonas	Colymycin or polymyxin B; streptomycin and terramycin or chloramphenicol occasionally may be effective
Proteus (indol negative) *E. coli—aerogenes*; paracolon	Cephalothin or ampicillin, or with streptomycin or occasionally with kanamycin; chloramphenicol in serious infections if the organism is sensitive
Mixed infections such as peritonitis	Penicillin or ampicillin and streptomycin; if there is penicillin allergy cephalothin and streptomycin or cephalothin alone

As indicated in table 2, the standard treatment for Klebsiella or Friedländer's pneumonia consists of streptomycin and one of the tetracycline group or chloramphenicol.[25, 26] Cephalothin or cephaloridine may be used as alternate therapy, since preliminary data indicate that they are effective if adequate dosage is employed.[27, 28] Pseudomonas infections are generally treated with Coly-Mycin® or polymyxin B. In the less severe infections in which the organism is sensitive a clinical response may occur with streptomycin and Terramycin®.

For Proteus, *E. coli* and paracolon infections in vitro sensitivity-testing is helpful in indicating the agent of choice. In these infections cephalothin or ampicillin is effective. Occasionally a combination of streptomycin and chloramphenicol may be required. For the indol-fermenting strains of Proteus (vulgaris, morganii and rettgeri), which are notoriously resistant to the antimicrobial agents, kanamycin may be used. In salmonella infections preliminary data suggest that ampicillin may be an alternative to chloramphenicol.

In mixed infections, such as in peritonitis, penicillin and streptomycin are the agents of choice. For patients allergic to penicillin, cephalothin or cephaloridine alone or with streptomycin is indicated. With mixed infections in the respiratory system and urinary tract, proper selection of an antimicrobial agent is generally dependent upon the sensitivity of the organisms isolated.

Since there are no fixed rules for combinations of antibiotics that will definitely be synergistic or antagonistic, the need remains for definitive bacteriology and in vitro sensitivity studies for the selection of the proper agent or combination of agents. It should be noted, however, that antibiotics such as chloramphenicol, lincomycin and erythromycin, all of which work at the 50 S ribosomal subunit, are potential competitors. The significance of these interactions, although not completely known, suggests that these antibiotics should not be employed simultaneously.

Method of Antibiotic Administration

The choice of the route of administration depends on the disease, the antimicrobial agent, and the condition of the patient. When the patient is in shock the intravenous administration of antibiotics produces the most consistent blood levels, hence the most effective tissue concentrations. When given intravenously, the antibiotic should be given in a relatively short period of time; 10 to 20 minutes is sufficient for penicillin, cephalothin, and lincomycin. For erythromycin, tetracycline, chloramphenicol and novobiocin, a 30- to 45-minute period is desirable.

In our judgment, for effective utilization antibiotics should not be given by constant intravenous drip over extended periods. In this connection, special attention should be given to the "piggyback" or tandem method of administration of antibiotics whereby the antibiotic passes through a second bottle of fluid during administration. We have seen instances in which the antibiotic has been greatly diluted by the second bottle without going directly to the patient.

Complications of Antimicrobial Therapy

Serious complications arising during the course of antimicrobial therapy are relatively infrequent when compared to the tremendous widespread use of these agents and the benefits derived. Nevertheless, such comparative statistics find little application in the treatment of the individual patient. Emphasis, therefore, must be placed on these reactions and their severity. This is not meant to disparage or discourage the proper use of the antibiotics, but rather to bring attention to the need for prevention of such complications and the necessity for the ever discriminating use of these potent agents. This following section reviews many of the more common side-effects of antimicrobial therapy.

The Penicillins (table 3). Untoward reactions to penicillin are generally limited to the hypersensitivity type, and pharmacologic toxicity does not occur with the usual therapeutic doses. The incidence of allergic reactions to penicillin has been estimated to range between 5 and 10 per cent.[29, 30] Sensitization is most frequent with topical or aerosol therapy, less frequent with parenteral therapy, and least by the oral route.

TABLE 3: Reactions to Antimicrobial Agents

- Penicillin:
  - Hypersensitivity—
    - Immediate (occurring within 30 minutes)
      - Anaphylaxis, particularly in patients with prior history of untoward reactions
      - Pseudo-anaphylaxis with procaine penicillin; absence of shock; patient psychotic, fearsome
      - Cutaneous reactions, usually urticarial, most common pattern; may appear as serum sickness
    - Accelerated (beginning within 48 hours)
      - Cutaneous reactions, urticarial, etc., as above
    - Late (occurring within 3 days or later)
      - Cutaneous reactions, urticarial, etc., as above; also erythematous-papulovesicular (may progress to exfoliation)
      - Drug fever, with and without eosinophilia with wide swings occurring during 12th and 25th days
      - Occur rarely—acquired hemolytic anemia, thrombocytopenia, variable renal involvement
  - Pharmacologic toxicity—
    - With rapid administration of massive I.V. doses (12-20,000,000 units)—C.N.S. irritability, convulsions and hyperreflexia; also cardiac arrhythmia and hyperkalemia occurring especially in patients with renal insufficiency
    - Crystalline penicillin G—
      - Potassium form: 1.5 mEq. K per 1 million units
      - Sodium form: 100 mg. Na per 1 million units
    - G.I.—Nausea, diarrhea, pruritus ani when given orally
    - Superinfection, secondary to overgrowth

A wide variety of allergic reactions occur,[31] and the immunologic mechanisms of penicillin allergy have been recently reviewed elsewhere.[32] Accordingly, allergic reactions may be classified as immediate (occurring within 30 minutes), accelerated (beginning within 48 hours), and late (3 days or later). Of the immediate types of reactions, anaphylaxis is the most serious and dramatic. This generally occurs in patients who have received penicillin previously and particularly in those with a history of untoward allergic reactions.

Special mention should be made regarding pseudoanaphylactoid reactions which occur immediately after the administration of procaine penicillin.[33, 34] The pattern of pseudoanaphylaxis due to procaine penicillin differs significantly from the true anaphylactoid reaction. One significant differential point is the absence of peripheral vascular collapse during pseudoanaphylactic reactions. The patient often appears acutely psychotic and frequently experiences an intense fear of impending death. There may be auditory and visual hallucinations in addition to other neuropsychiatric symptoms. It should be appreciated that the prevention of self-inflicted injury during this reaction is of the utmost importance.

Cutaneous manifestations, usually urticarial, are the most common allergic reactions to penicillin and may be part of the immediate, accelerated, or late type of reactions. As such, they can occur with or without systemic manifestations, i.e., angioneurotic edema or serum-sickness-like syndrome. Other delayed manifestations include drug fever, acquired hemolytic anemia,[35, 36] thrombocytopenia,[37] and varying degrees of renal involvement.[38, 39]

Although pharmacologic toxicity does not arise with the usual therapeutic doses of penicillin, difficulties may arise under certain circumstances. This has been particularly true when massive intravenous doses (20 or more million units per day) are given rapidly. Weinstein and colleagues[40] have noted the occurrence of C.N.S. irritability with convulsions, cardiac arrhythmias, and hyperkalemia with massive penicillin therapy, particularly in patients with renal insufficiency. It should be appreciated that in patients with renal insufficiency or congestive heart failure electrolyte complications may result because there are roughly 1.5 mEq. of potassium per million units of penicillin and approximately 100 mg. of sodium per million units if the sodium salt is used.

All patients should be carefully questioned regarding previous adverse reactions to penicillin and any type of allergic condition in their history.

The Tetracyclines (table 4). The tetracycline group of antibiotics appear to be relatively safe, significant untoward effects other than gastrointestinal problems being infrequent. Three of the

4 commonly employed tetracyclines are similar in their therapeutic, pharmacologic, and toxicologic properties. Tetracycline (TC) seems to be less inclined to produce untoward effects than oxytetracycline (OTC), chlortetracycline (CTC), or demethylchlortetracycline (DMCT).[41, 42] None of the congeners seem to provide therapeutic superiority, although at times minor differences in antibiotic sensitivity patterns may be observed.

TABLE 4: Reactions to Antimicrobial Agents

- Tetracyclines:
  - Toxicity—
    - G.I. symptoms most common (cheilitis and glossitis may respond to vitamins)
    - Superinfection—serious problem for long-term therapy, staphylococcal enteritis, pseudomembranous colitis, candida
    - Photosensitivity—most common with demethylchlortetracycline
    - Discoloration of teeth in children and infants of mothers given prolonged therapy. Deposition in bones.
    - Hepatic—fatty liver, jaundice with high dose I.V. therapy; negative nitrogen balance; increased riboflavin in urine
    - Adult Fanconi syndrome—outdated preparations
- Chloramphenicol. Use only in serious infections as an alternate or as indicated by laboratory and clinical evaluation
  - Toxicity—
    - Hematopoietic. May cause aplastic anemia and other blood dyscrasias; increased incidence with renal and hepatic disease; reticulocytes decreased, serum iron increased. Bone marrow—vacuolization pronormoblasts.
    - Gray disease in infants

The major complications of tetracycline therapy are limited to the gastrointestinal tract and include nausea, vomiting, and diarrhea. The diarrhea is generally self-limited after the cessation of oral therapy; however, superinfection with staphylococci or Candida may be serious. Diarrhea appears most frequently after OTC and CTC and occurs about 4 per cent of the time with TC.[41, 42]

Severe hepatotoxicity, consisting of extensive fatty infiltration of the liver, is uncommon. The early studies by Lepper and colleagues[43] indicated that large doses of CTC, given orally and intravenously, produced serious hepatic dysfunction. Subsequently, it was demonstrated that, although large doses of any of the tetracyclines might induce similar changes, CTC was the most toxic.[44]

Over the past several years attention has been directed toward the occurrence of fatal hepatotoxicity induced by tetracycline therapy during pregnancy.[45-48] Most of these patients had been treated for pyelonephritis, frequently with excessive doses by the intravenous route. Their clinical courses were marked by nausea, vomiting, fever (often with wide fluctuations in temperature), and icterus, and terminally by hematemesis, azotemia, and acidosis. Extensive fatty infiltration of the liver has been found at autopsy.

Other untoward effects of tetracycline therapy have been noted in infants of mothers receiving tetracycline during the late stages of pregnancy. Included are disturbances of odontogenesis with discoloration of the teeth[49, 50] and the deposition of tetracycline in bone as well as interference in bone growth.[51]

In view of these serious untoward effects, it would appear that the use of tetracyclines should be limited during pregnancy and infancy.

Abnormalities of renal function with negative nitrogen balance may occur with any of the tetracyclines,[52, 53] and renal tubular damage has resulted from outdated preparations,[54, 55, 56] as well as from prolonged usage of DMCT.[57] The syndrome of temporary renal dysfunction produced by outdated preparations includes glycosuria, amino-aciduria, phosphaturia, hypokalemia, and proteinuria. Acidosis and azotemia also occur. Recently a nephrogenic diabetes-insipidus-like syndrome has been observed after prolonged therapy with demethylchlortetracycline.[57]

Other untoward effects of tetracycline therapy include photosensitization which appears most commonly with DMCT[58, 59] and the occurrence of peptic ulcerations in patients with severe renal disease.[60] Benign intracranial hypertension, although rare in adults,[61] is more frequently recognized in infants because of the presence of bulging fontanelles.[62] Allergic reactions to the tetracyclines are most infrequent, but anaphylaxis has been reported.[63, 64]

Chloramphenicol (table 4). Chloramphenicol is an effective broad-spectrum antibiotic which is generally well tolerated. The unfortunate occurrence of significant hematologic complications, particularly aplastic anemia and less frequently agranulocytosis and/or thrombocytopenia, precludes its casual usage. The exact incidence of severe or fatal aplastic anemia is unknown, but estimates of 1:10,000 to 1:100,000 patients treated seem most reasonable.[65, 66]

Although there is no way to predict which patient will develop hematologic complications, bone marrow depression occurs frequently when the chloramphenicol blood level reaches 25 micrograms per ml. or more.[67] Accordingly, it has been suggested that blood levels in excess of 15 to 20 micrograms per ml. provide little therapeutic advantage, since most chloramphenicol-sensitive microörganisms will respond to levels that are significantly less.[67] It would appear that the majority of hematologic toxicity is dose-related, but individual susceptibility is also a factor. This is suggested by the higher incidence of hematologic complications occurring in children and in females.[68, 69] In addition, patients with hepatic and renal disease are seemingly more prone to these untoward effects.[70]

Another potential complication of chloramphenicol therapy, the "gray syndrome," occurs in premature and newborn infants in whom large doses have been employed. Under these circumstances both the free and the conjugated levels of chloramphenicol are greatly elevated. This has been related to the "metabolic immaturity" of the liver, which normally conjugates chloramphenicol, and to the decreased excretion by the kidney.[71]

Other untoward effects include mild gastrointestinal symptoms, usually diarrhea or stomatitis, peripheral neuritis, and optic neuritis[72] after prolonged treatment, and rarely allergic reactions. Recent observations suggest that chloramphenicol is capable of suppressing the anamnestic response to tetanus toxoid.[73]

Erythromycin (table 5). Erythromycin is undoubtedly one of the least toxic of all the antimicrobial agents employed today.[74, 75] Adverse effects are generally limited to gastrointestinal irritability, and allergic reactions are extremely rare.[76]

Erythromycin is currently available in a number of different forms, each differing in their absorption and toxicity. A film-coated stearate salt (Erythrocin®) and an acid-resistant erythromycin base (Ilotycin®) are less well absorbed from the gastrointestinal tract than the estolate salt (Ilosone®).[77] On the other hand, the estolate salt may infrequently produce hepatic dysfunction after 10 to 14 days of oral administration.[78, 79, 80] Other symptoms include nausea, vomiting, and abdominal cramps early, followed by fever, icterus, and eosinophilia, as well as abnormalities of hepatic function.[80] This is believed to be a hepatosensitivity reaction with an estimated incidence of 2 to 4 per cent.[81, 82]

Streptomycin (table 6). Streptomycin provides one of the cornerstones of combined antibiotic therapy, and thus the indications for its use are restricted.

The majority of untoward effects of streptomycin therapy are of 2 types—neurotoxicity and hypersensitivity. The major neurotoxic effect consists of damage to the vestibular mechanism,[83] although auditory acuity may also be affected.[84] Symptoms including tinnitus, nausea, vomiting, vertigo, ataxia and circumoral paresthesias may be noted early. Another neurotoxic effect produced by streptomycin is a curare-like neuromuscular blockade. Several patients have been reported to have developed a myasthenic syndrome which responded to neostigmine and cessation of streptomycin therapy.[85] Instances of prolonged respiratory paralysis after intraperitoneal administration have also been reported.[86]

TABLE 5: Reactions to Antimicrobial Agents

Erythromycin (stearate and base)	Occasional abdominal cramping and diarrhea in ambulatory patients. Estolate salt may produce hepatic dysfunction after 10 to 14 days
Novobiocin	Cutaneous reactions; occasional transient leukopenia; yellow discoloration of skin (chromogenic metabolite); hepatic dysfunction occasionally
Oleandomycin (triacetyl)	Hepatic dysfunction frequently by 7 to 14 days; cutaneous reactions
Lincomycin	Orally, G.I. symptoms, less if administered 1 hour a.c. or p.c.
Cephalosporins	Hypersensitivity may occur, cutaneous primarily
Cephalothin	Discomfort I.M.
Cephaloridine	Well tolerated I.M.; possible nephrotoxicity above 4 to 6 Gm. daily. Decrease dose with renal failure.
Isoniazid	Peripheral neuropathy (from high dosage, and in patients who are slow inactivators)—mental disturbance, convulsive disorders; optic neuritis; pyridoxine indicated concurrently
PAS	G.I. symptoms, hypersensitivity, fever, skin rash, lymphadenopathy; occasional infectious mononucleosis-like syndrome but negative heterophile; occasional hepatic dysfunction

A variety of hypersensitivity reactions to streptomycin may occur.[84] These include the usual allergic cutaneous reactions as well as contact dermatitis, drug fever, eosinophilia and, less commonly, angioneurotic edema and acute anaphylaxis.

Nephrotoxicity to streptomycin is uncommon[87] but may occur when combinations of nephrotoxic antibiotics are used concurrently.[88]

Kanamycin, neomycin and paromomycin (table 6). All 3 of these antibiotics which have been obtained from soil actinomycetes will be considered together because of their similarities. Neurotoxicity and nephrotoxicity are the most frequent and severe side-effects.[89] Although kanamycin is the only one generally employed for parenteral use, it should be appreciated that more pronounced toxicity occurs under similar conditions with neomycin.[90, 91] Because of its toxicity, paromomycin is limited strictly for oral use in this country.

TABLE 6: Reactions to Antimicrobial Agents

Streptomycin	Neurotoxicity, vestibular mechanism—auditory occasionally; curare-like neuromuscular blockade (myasthenic syndrome) (responds to neostigmine and cessation of streptomycin); cutaneous hypersensitivity
Kanamycin	May be nephrotoxic and ototoxic, particularly with more than 1 Gm., or prolonged treatment, or renal insufficiency; avoid with other nephrotoxic agents
Neomycin	Nephrotoxic and ototoxic by injection of more than 1 Gm. per day, and orally in presence of renal insufficiency; superinfection, with Staphylococcus or Candida Intraperitoneal instillation of kanamycin or neomycin may produce neuromuscular blockade. Calcium can reverse paralysis.
Polymyxins	Daily maximum—poly B, 2.5 mg./Kg, colymycin (poly E, 5 mg./Kg). Neurotoxicity—paresthesias; occasionally ataxia and vertigo may interfere with neuromuscular transmission. Respiratory arrest may occur with high serum concentration.
Vancomycin	High blood concentrations may be ototoxic. Caution with impaired renal function; occasionally hypersensitivity
Amphotericin B (antifungal)	Possible anorexia, chills, fever. May be nephrotoxic. In prolonged therapy hepatic and hematologic studies indicated.

N.B. With the above decrease dose with renal failure.

A recent symposium discusses many of the important clinical, pharmacologic and toxicologic aspects of kanamycin.[92] Neurotoxicity consists mostly of auditory-nerve damage, although vestibular impairment may occur. Early ototoxicity loss at the higher sound frequency has been observed by audiometric studies.[93] It has been suggested that tinnitus often appears before significant loss in the spoken-voice range develops, but abrupt loss of hearing frequently occurs without significant warning.[89] Pathologically, ototoxicity has been correlated with the destruction of the outer hair cells of the cochlea.[94] This is almost always bilateral and irreversible. Clinically, the incidence of toxicity is directly related to the total dose, duration of therapy, age, and renal function.

Another potentially serious effect produced by this group is neuromuscular blockade. A few clinical reports indicate that this occurs quite rapidly after the intraperitoneal instillation of neomycin or kanamycin.[95, 96] Similar effects may occur after either intraperitoneal or intrapleural instillation of any of these agents, but neomycin appears to be the most potent offender. Unlike streptomycin or neomycin, kanamycin cannot be antagonized by neostigmine.[97] Conversely, d-tubocurarine may potentiate the blockade produced by all 3 of these antibiotics, and calcium ion is capable of reversing the paralysis caused by this group.[97]

Nephrotoxicity is a potential hazard with these antibiotics and is usually reversible.[89] The resultant renal insufficiency, although of singular significance, is often compounded by the fact that it predisposes to ototoxicity which is frequently permanent. The detection of abnormalities in urinary sediment, particularly red blood cells and granular casts, may be the harbinger of significant renal histologic damage and functional impairment.

The combination of kanamycin with other antibiotics, particularly those with potential nephrotoxic effects, should be avoided. This is true with the polymyxins, since they might be used in combinations where Pseudomonas species is suspected.

Other side-effects relate mainly to oral therapy and include gastrointestinal complaints, the most serious of which may be diarrhea and superinfection with staphylococcus or Candida. Care should also be taken in patients with preëxistent renal failure because accumulation of these poorly absorbed drugs may occur.

Vancomycin (table 6). Vancomycin, although seldom used because of the availability of the new and potent antistaphylococcal agents, has been extremely successful in the treatment of serious systemic staphylococcal infections.[98, 99] Despite the newer replacements it is well worth remembering that virtually all the staphylococci are sensitive to low concentrations of this antibiotic (1 to 2 micrograms) and that only rarely have these microörganisms been truly resistant.

Ototoxicity and nephrotoxicity are the major untoward effects.[100, 101] Excessively high doses and impairment of renal function predispose to this toxicity, since excretion is wholly dependent upon glomerular filtration. Recent observations in oliguric patients suggest that a single intravenous dose of 1 Gm. of vancomycin may produce therapeutic blood levels for as long as 10 to 14 days.[102]

Other significant untoward effects include allergic skin eruptions, fever, chills, and occasionally vascular collapse. Most frequently, however, is the occurrence of local thrombophlebitis at the site of intravenous administration.[100]

Polymyxins (table 6). Recent concern over the increasing incidence of serious gram-negative infections, particularly *Pseudomonas aeruginosa*, has resulted in more widespread use of the polymyxins (polymyxin B, colistin-polymyxin E). The major side-effects are neurotoxicity and nephrotoxicity. Circumoral paresthesias occur most often, although ataxia, vertigo, and other sensory changes have been noted.[103] The polymyxins can also interfere with neuromuscular transmission.[104] Respiratory arrest has been noted to occur, particularly when high serum concentrations have been attained.[105, 106]

Nephrotoxicity, although not an uncommon effect, is generally reversible.[107] Proteinuria and abnormal urinary sediment may presage the onset of azotemia. Despite this, therapy may be continued with decreased doses in face of renal failure.[108] Other side-effects have been uncommon, and allergic reactions are rarely encountered.

Lincomycin (table 5). Lincomycin is a clinically effective antibiotic which was first isolated from the fungus *Streptomyces lincolnensis* in 1962.[109] Although chemically distinct from the other known antibiotics, it possesses a range of activity comparable to that of erythromycin for gram-positive microörganisms[110] and has activity against staphylococcal L-forms.[111] Lincomycin appears to be relatively nontoxic, and there is no evidence of cross allergenicity with any of the known antimicrobial agents.[112] Mild to moderately severe diarrhea occurs with the oral preparation, but this may be minimized by the administration in a fasting state or 1 hour before a meal. An increase in the number of enterococci has been observed in patients with diarrhea induced by lincomycin. It should be noted also that oral lincomycin therapy may effect an increase in Pseudomonas species and Candida in the stools and a corresponding reduction of the anaerobic flora.[113] Other gastrointestinal side-effects include occasional nausea, vomiting, and abdominal cramps. Other untoward effects are unusual, although a skin rash may rarely be encountered.

Cephalosporins (table 5). Cephalothin and a newer analogue, cephaloridine, represent a new class of antibiotics which are clinically effective against a wide variety of gram-negative bacilli and gram-positive cocci (including resistant staphylococci).[114, 115] Both appear to have a low incidence of untoward effects, and preliminary studies have suggested a lack of cross sensitivity with the penicillins. This latter point seems to be under question, since there have been reports to the contrary.[116, 117] Nevertheless, many patients who have documented penicillin allergy have received cephalothin or cephaloridine without untoward effect.

Cephalothin is frequently painful when given intramuscularly and may produce thrombophlebitis at the site of intravenous infusion.[118] Urticaria and a variety of cutaneous reactions may occur with both antibiotics.[119] Nephrotoxicity has been reported in patients receiving large doses of cephaloridine (generally more than 6 Gm. per day).[120] This should not be a significant problem, since it is unlikely that sensitive microörganisms will require this dosage.

Hematologic effects noted during cephalothin therapy may include Coombs' positive reactions,[121] and there have been 3 instances of leukopenia.[122] Eosinophilia has been observed alone, as well as with cutaneous and systemic reactions.[119]

Amphotericin B (table 6). Amphotericin B, a polyene antibiotic, is the only effective parenteral antifungal agent for treating disseminated histoplasmosis, cryptococcus, and coccidioidomycosis infections.

A myriad of adverse reactions occur with intravenous therapy. These include toxic and hypersensitivity phenomena. Anaphylaxis may be observed, but the usual untoward effects are chills, fever, headaches, nausea, and emesis. These reactions may be minimized by antihistaminics or when of a more severe nature by administering 25 to 100 mg. of hydrocortisone simultaneously with the amphotericin. Thrombophlebitis occurs frequently.[123, 124] Although these symptoms are more acutely troublesome to the patient, the uniform deterioration of renal function,[125] progressive hemolytic anemia, and electrolyte abnormalities, particularly hypokalemia, represent a significantly greater problem.[123] Hypomagnesemia,[126] renal tubular acidosis,[127] and nephrocalcinosis have also been reported.

Various forms of neurotoxicity ranging from tremors to paralysis of the extremities and accompanied by incontinence of urine and feces have been observed.[128] As with the myositis[129] and possible respiratory paresis, many of these manifestations may be related to acute electrolyte shifts rather than to direct toxic effects.

Griseofulvin. Griseofulvin, an oral fungistatic antibiotic produced by many strains of penicillia, has been used successfully in the treatment of various superficial fungal infections. Although low concentrations of griseofulvin inhibit the growth of many superficial dermatophytes,[130] the duration of therapy is often quite long, being a function of many factors including the site and extent of the lesion.

In general, griseofulvin has been a relatively safe drug with infrequent side-effects.[131] These range from mild gastrointestinal complaints to headaches, paresthesias, fatigue, and insomnia. In addition to the usual cutaneous drug reactions, photosensitivity has also been observed.

A number of interesting "nonfungal" effects have been noted during the course of therapy with griseofulvin.[131] These include derangements of porphyrin metabolism, with exacerbations of porphyria,[132] possible anti-inflammatory action, and reported anti-anginal effect in patients with coronary artery disease.[131]

The Sulfonamides. The sulfonamides continue to enjoy wide usage despite the fact that there are relatively few situations in which they are the agents of first choice. The availability of a large number of more effective antimicrobial agents with less untoward effects would appear to limit their clinical usefulness. Sulfonamides are no longer considered to be the agents of choice for meningococcal infections because of the prevalence of sulfadiazine-resistant sero-group B meningococci.[133]

The precise frequency of adverse reactions to the sulfonamides is unknown, and estimates range from 5 to 10 per cent. Serious reactions probably occur in less than 1 per cent of the patients treated. Many variables such as age, type of sulfonamide used, dosage, state of hydration, allergic diathesis, and enzymatic defects play a role. With the exception of kernicterus in the newborn,[134] acute hemolytic anemia,[135] and the Stevens-Johnson syndrome,[136] children appear less inclined to develop untoward reactions.[135]

The recent experiences with the long-acting sulfonamides, sulfamethoxypyridazine (Kynex®, Midicel®) and sulfadimethoxine (Madribon®), suggest that they have a greater frequency of serious untoward effects.[137] Well over 100 cases of Stevens-Johnson syndrome,[137] as well as a related condition, toxic epidermal necrolysis, have been attributed to the long-acting sulfonamides.[138] The majority of these reactions have been in children, with a resultant high mortality rate. Significantly, attention has been called to the fact that the long-acting sulfonamides have been excluded from the British National Formulary.[136] In this country the short-acting preparations will suffice for all circumstances in which the long-acting preparations might be employed.[137]

The long-acting sulfas have been demonstrated to bind plasma proteins to a greater extent and have resulted in prolonged blood levels in the newborn of mothers receiving these agents before delivery.[139] Because these drugs compete with, and may displace bilirubin from binding sites of the plasma proteins, kernicterus has resulted with lower than usual toxic bilirubin levels.[140] Other sulfonamides, particularly sulfisoxazole, can also produce this phenomenon. It would seem prudent to avoid the use of sulfonamides, particularly the long-acting variety, during the latter part of gestation.

Serious nephrotoxicity is usually the result of sulfonamide crystals precipitating in acid urine, particularly if the urine volume is meager.[141] Combinations of sulfonamides and mandelamine may form insoluble materials which plug the renal tubules and result in acute renal insufficiency.[142]

Other untoward effects include various hypersensitivity reactions, drug fever, bizarre neurologic syndromes, pancreatitis, and sulfonamide-induced hepatitis.

Almost all varieties of hematologic side-effects have been reported, and hemolytic anemia occurs frequently in susceptible individuals with G6PD deficiency. Agranulocytosis, thrombocytopenia, and methemoglobinemia have also been noted.[138]

Isoniazid (INH) and para-aminosalicylic acid (PAS) (table 5). INH and PAS produce a number of adverse reactions, many of which are minor but nevertheless disturbing to the patient. At low dosage (5 mg./Kg) INH has proved to be a useful and relatively safe drug. Peripheral neuropathy has been observed more frequently with higher dosage and particularly among patients who are slow inactivators of INH. Mental disturbances, convulsive disorders, optic neuritis, and encephalopathy have also been described.[143] The routine administration of pyridoxine seems to be of value, particularly with prolonged therapy or when larger doses of INH are used.[144] Other side-effects include gastrointestinal symptoms, anemia, hepatitis, and rarely an infectious mononucleosis-like syndrome.[145] A pellagra-like syndrome which is pyridoxine-responsive has also been observed.[143]

PAS is less well tolerated than INH and results in a greater frequency and variety of adverse reactions. Toxic side-effects include gastrointestinal discomfort, nausea, vomiting, and diarrhea.[146] These symptoms may be lessened to some degree by changing to another preparation such as the calcium salt. Frequently, beginning with small daily doses and gradually increasing them results in better tolerance.

Hypersensitivity reactions are common and include fever, skin rash, gastrointestinal symptoms, and often lymphadenopathy. Rarely an infectious mononucleosis-like syndrome may occur and is only distinguishable by a negative heterophile and reappearance of symptoms after challenge with even small doses of PAS.[143] Potentially serious hepatic dysfunction has been noted frequently in these patients with hypersensitivity reactions. Zimmerman[81] has estimated that about 25 per cent of the patients having reactions to PAS also exhibit jaundice and that the mortality rate may be as high as 10 per cent in this group.

Other untoward effects include hemolytic anemia, transient pulmonary infiltrates, acute psychotic reactions, encephalitis, goiter with or without hypothyroidism, and hypokalemia.[143]

Other Antimicrobial agents (table 5). Several other antibiotics and chemotherapeutic agents are known to produce frequent adverse reactions and will not be discussed in great detail because they offer little therapeutic advantage. Both oleandomycin and triacetyloleandomycin produce gastrointestinal symptoms, skin rash, and drug fever. The latter antibiotic frequently produces hepatic dysfunction and jaundice[147] and should not be given for more than 7 to 10 days. Indications for the use of these agents appear very limited since they offer little if any advantage over any of the currently available antibiotics of a similar spectrum.[148]

Novobiocin may produce cutaneous reactions in about 10 per cent of patients.[149, 150] Drug fever, eosinophilia, and gastrointestinal complaints with mild diarrhea occur. Hemolytic anemia and leukopenia are rare. A peculiar yellow discoloration of the skin, sclerae and serum has been noted and is probably due to a chromogenic metabolite of novobiocin.[149, 151] Hepatic dysfunction has been noted, because novobiocin inhibits glucuronyl transferase[152] and has been associated with neonatal jaundice.

Nitrofurantoin has been widely used for the treatment of urinary-tract infections. Negligible blood levels occur after oral therapy, and consequently this drug should never be employed for systemic infections. Gastrointestinal side-effects include nausea and vomiting which often require discontinuation of therapy.[153] There have been an increasing number of reports of peripheral neuropathy.[154] Impaired renal function has been present in almost all these instances and the blood levels of nitrofurantoin found to be considerably elevated.[154] Other side-effects include hemolytic anemia in patients with G6PD deficiency, and rarely pulmonary infiltrates and eosinophilia.[155] It is suggested that nitrofurantoin not be given during pregnancy.[156]

Conclusion

We have discussed a number of principles and factors which have influenced the trend in antimicrobial therapy and represent an expansion of those set forth in an earlier paper.[157] It has not been our intention to minimize the importance and effectiveness of the wide variety of antimicrobial agents but to pinpoint the necessity for definitive microbiology and diagnosis as well as to place emphasis on the untoward effects which frequently are unrecognized. With the number of antimicrobial agents at our command one can tailor antimicrobial needs specifically and effectively, and we hope that the principles and factors enunciated will lead to a discriminate use of these potent agents.[158]

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Leukemia in Pregnancy

Data on Nine Cases With an Important New Finding

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PREGNANCY complicated by leukemia is infrequent. Moloney[1] counted 267 cases in the world literature. A review of the literature prior to 1943 was published by McGoldrick and Lapp.[2] They presented statistics on 75 cases of acute or chronic leukemia in pregnancy. Summaries of the literature before 1943 also include those by Grier and Richter,[3] Forkner,[4] Mehta,[5] Garrasi,[6] Debiasi,[7] Neumann,[8] Bower and Clark,[9] Ohlsson,[10] and Kosmak.[11] Yahia, Hyman and Phillips[12] reviewed the literature on acute leukemia and pregnancy for the period of 1943 1956. The combination of acute leukemia and pregnancy has also been reviewed by Erf, [13] Frenkel and Meyers,[14] Lee and his colleagues,[15] and Ask-Upmark.[16] Sheehy[17] also tabulated his summary on chronic leukemia in 1957. Results of all the reported cases of acute leukemia and pregnancy from 1954 through 1964 were compiled by Hoover and Schumacher.[18]

During the past 10 years some of the contributors of more than 2 cases of leukemia in pregnancy include Guasch[19] (5 cases, 1962), Raichs[20] (4 cases, 1962), Jaluvka[21] (10 cases, 1962), Rothberg, Conrad and Conley[22] (4 cases, 1959), Lee, Johnson and Hanlon[15] (12 cases, 1962), Wiedermann et al[23] (7 cases, 1956), Myasnikov[24] (8 cases, 1963), and Boggs, Wintrobe and Cartwright[25] (5 cases, 1962).

During the past 4 years there have been at least 37 cases of leukemia and pregnancy reported in the literature.[1, 17, 19, 25-45] These represented 7 cases of acute and 17 cases of chronic granulocytic leukemia, 6 cases of acute and 1 case of chronic lymphocytic leukemia, 1 case of acute monocytic leukemia, and 5 cases of unspecified acute leukemia.

Pertinent data of the 9 cases of leukemia in pregnancy recorded at the George Washington University Hospital and the District of Columbia General Hospital, Washington, D. C., during the past 13 years are presented in tables 1-A, 1-B and 1-C. The tabulation is an attempt to standardize some of these criteria and follows somewhat the format in previous reviews of the literature on this subject. It is recommended that future reports of such cases record at least the 14 major categories outlined in this presentation. There will be, no doubt, additional pertinent data that will need to be added as we learn more about leukemia in pregnancy.

Discussion

The incidence of leukemia in pregnancy is difficult to estimate. The annual death rate in women in the child-bearing age group in general is estimated at 1 in 100,000. "On that basis one would expect less than 1 case of acute leukemia in 75,000 deliveries. . . .[12] The estimates from our series place this figure at 1 in 14,000.

Any suggestion of an increase in incidence is debatable from many viewpoints. It could be due to the critical prolongation of life afforded by the newer antileukemic therapy. The important realization is that leukemia in the pregnant patient is not more infrequent than in the nonpregnant female as was previously thought.

Regardless of the exact incidence, all clinicians are interested in its early diagnosis during pregnancy, in its treatment, and in the prediction of the course of the disease in the mother as well as the outcome of the fetus. These objectives can be achieved by keeping accurate records in the literature.

The mean age of expectant mothers with acute leukemia was 27 years, with a range from 18 to 31 years. This finding closely agrees with those reported by other investigators.[2, 12, 18] There was 1 case of chronic leukemia (patient aged 28). The disease occurred more frequently in Caucasian women than in Negro women. Of almost 46,000 deliveries to mostly Caucasian women (98 per cent) at one hospital there were 5 Caucasian and 1 Negro patients, while at another hospital at which 98 per cent Negro babies were delivered only 3 cases (all Negro) were found among 79,000 deliveries. Since the former group received much

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TABLE 1-A: Data on 9 Cases of Leukemia in Pregnancy at George Washington University and D. C. General Hospitals, 1955–1967

Case and Year	Age	Race	Gravida and Para	Onset	Earliest Signs and Symptoms	Leukemia Classification
1. F. M. 1958	27	Negro	G II, P 1	2 months prior to conception	Fatigue, dizziness, swelling of gingivae, adenopathy	Acute granulocytic
2. J. N. 1964	26	Caucasian	G IV, P 3	Beginning third trimester	General weakness, adenopathy	Acute granulocytic (aleukemic)
3. J. S. 1959	28	Caucasian	G II, P 1	First trimester	Dyspnea, fatigue, chest pain, palpitation, ankle edema, adenopathy	Acute lymphocytic
4. B. R. 1954	31	Caucasian	G VI, P 1, ab 5	Third trimester	Severe gingivitis, easy bruising	Acute monocytic
5. D. C. 1958	24	Caucasian	G II, P 1	First trimester	"Severe infection" secondary to cat scratch, splenomegaly, adenopathy	Acute lymphocytic
6. A. W. 1967	27	Caucasian	G II, P 1	1 month prior to conception	Weakness, splenomegaly, epistaxis	Acute granulocytic
7. M. H. 1964	28	Negro	G V, P 4	Late third trimester	Splenomegaly	Chronic granulocytic
8. B. B. 1959	19	Negro	G II, P 1	Second trimester	Bleeding gums, petechiae over arms, legs and face, "dark-colored" stools and urine	Acute monocytic
9. L. G. 1957	18	Negro	G II, P 0, ab 1	First trimester	Weakness, anorexia, bleeding gums	Acute lymphocytic

TABLE 1-B: Data on 9 Cases of Leukemia in Pregnancy at George Washington University and D. C. General Hospitals, 1955–1967

Type of Delivery or Abortion	Fetal Outcome and Weight	Weeks of Gestation	Maternal Hemorrhage	Thrombocytopenia
1. Spontaneous abortion		4	Required 12 units whole blood for 10 days postpartum	Yes
2. Normal spontaneous delivery	Apparently healthy; 8 lbs. 14 oz.	38	Within normal limits	Yes
3. Normal spontaneous delivery	Premature; 3 lbs.	34	Within normal limits	Yes
4. Mid forceps	Apparently healthy; 5 lbs. 14 oz.	38	Within normal limits	Yes
5. Stillborn	Appeared shrunken	28 or more?	Yes—transfused 2 units whole blood	Yes
6. Therapeutic abortion		20	Yes—transfused 4 units whole blood	Yes
7. Normal spontaneous delivery	Apparently healthy; 7 lbs. 6½ oz.	39	Yes—transfused 4 units whole blood	Yes
8. Low forceps	Died 1 hour after birth; 1 lb. 12 oz.	28	Yes—transfused 2 units whole blood	Yes
9. Spontaneous abortion		8	Yes—transfused 2 units whole blood	Yes

better prenatal care than the latter, one could infer that the poor prenatal care had little significance in the etiology of leukemia in these patients.

McGoldrick and Lapp[2] as well as Allen[46] suggested that most of the leukemic pregnant patients were primigravidas or primiparas, whereas our study showed the mean gravidity to be 2 and the parity to be 1.

TABLE 1-C: Data on 9 Cases of Leukemia in Pregnancy at George Washington University and D. C. General Hospitals, 1955–1967

Blood Group and Rh	Maternal Outcome	Duration of Disease	Treatment during Pregnancy	Remarks
1. A-positive	Died 1 month after abortion	1 yr.	Transfusions, corticosteroids	
2. A-positive	Died 4 days postpartum	3 mo.	6-Mercaptopurine, transfusions	Infectious mononucleosis had been diagnosed 5 yrs. previously
3. A-positive	Died 3 weeks postpartum	5 mo.	6-Mercaptopurine, transfusions, corticosteroids	⅓ of placenta infarcted
4. A-positive	Died 2 days postpartum	2 mo.	6-Mercaptopurine, transfusions	Patient had congenital absence of gall-bladder
5. A-positive	Died 1 day after abortion	6 mo.	6-Mercaptopurine, corticosteroids	Previous child had clubfoot
6. A-positive	2-month remission without drug therapy after abortion; then died	7 mo.	6-Mercaptopurine, corticosteroids, vincristine, transfusions	
7. A-positive	Lived 2 years after delivery	2 yrs.	None	One report of lymph node biopsy showed lymphosarcoma
8. O-positive	Died 9 days after delivery	2 mo.	Transfusions, corticosteroids, 6-mercaptopurine	
9. O-positive	Died 2 months after abortion	4 mo.	Transfusions, corticosteroids, 6-mercaptopurine	

The onset of acute leukemia was equally common during first and third trimesters and in all but 2 cases had its onset during pregnancy.

Generalized fatigue, dizziness, inflammation and swelling of the gingivae, dyspnea on exertion, petechiae, purpura, splenomegaly, and adenopathy were among the most common initial symptoms and signs.

Our data differ considerably from those in most of the literature in regard to the stated 2 to 1 ratio of chronic to acute leukemia in pregnancy. Only 1 case in 9 was chronic granulocytic leukemia. Moloney[1] noted an increase of acute granulocytic leukemia and a decrease of chronic granulocytic leukemia. The overall increase in incidence of acute leukemia has also been discussed by Boggs et al.[25] The predominant type of acute leukemia appears to be granulocytic (46 per cent) according to Hoover and Schumacher.[18] In our study acute granulocytic leukemia equaled that of acute lymphoblastic leukemia. In addition there were 2 cases of acute monocytic leukemia.

The type of delivery was usually normal spontaneous, but premature labor is higher in all series reported. Even though many babies are born prematurely, the neonatal mortality has not been appreciably higher in babies born to chronic leukemic mothers. Fetal mortality during pregnancy and parturition has always been higher when the mother had acute leukemia and furthermore has not changed significantly in the last 5 years (table 2).

Maternal postpartum hemorrhage may be a serious problem in the patient with acute leukemia. McGoldrick and Lapp[2] reported 3 fatal hemorrhages in 75 cases. It is usually related to thrombocytopenia, although hypofibrinogenemia could be the more important factor because of its occult nature. Thrombocytopenia was present in all patients. Six out of 9 had excessive hemorrhage problems. Fibrinogen levels or other coagulation factor deficiencies were not analyzed in any of the patients and thus remain as clinically important investigative goals.

An interesting new finding which has not been reported in the literature is that almost 80 per cent of the pregnant patients with leukemia in our study had A-positive ("D") blood type. At present it is impossible to incorporate this finding into any theories of pathogenesis or the course of the disease. It could be that the A-positive female is constitutionally more susceptible to leukemia during pregnancy, or that there is a change or loss of a blood group antigen (*vide infra*). Statisticians would favor attaching significance to all such results. Thus one can only declare that future investigations will have to be made in order to ascertain the real value of this observation.

TABLE 2: Fetal and Maternal Mortality during Pregnancy and Parturition*

	Type of Leukemia in Mother	To 1943 Per cent	1943–1956 (1947–1957) Per cent	1958–1963 Per cent	1963–1967 Per cent	1955–1967 (this study) Per cent
Fetal	Chronic granulocytic	16	7.6	13.6	12	0
Maternal	Chronic granulocytic	7	0	0	0	0
Fetal	Acute	60	38.7	34	33	25
Maternal	Acute	29	20	11.6	11	12.5

* Supplemented and modified after Moloney[1] and includes deaths within 24 hours of delivery.

Hyman and his colleagues[47] described 21 cases in 1963 in which carcinoma of the colon developed in association with leukemia or lymphoma and a disproportionately high percentage of their patients (65 per cent) had group A blood type, with 18 of 20 patients having D-positive Rh group. It has also been reported[48] that in patients with multiple primary neoplasms there is a striking excess of those with blood group A. From Clark's review[49] it appears that in cases with gastric carcinomas, pernicious anemia, diabetes mellitus, cancer of the pancreas and cancer of the esophagus there is an excess of patients of group A. Tumors of the salivary glands have similarly been associated with group A patients.[50, 51] Finally, Richards[52] described the loss of blood group B antigen in an AB type patient with chronic lymphocytic leukemia, thus changing his group from AB to A.

Maternal mortality is compared in table 2. The low mortality in chronic granulocytic leukemia has been challenged by some investigators. Few[3, 53] have contended that pregnancy is harmful in leukemia and may shorten the life span or precipitate a "blastic crisis." The pregnancy seemed to have aggravated all 8 of Myasnikov's leukemic patients, since all died within 1 month after delivery.[24] He concluded that pregnancy was absolutely contraindicated in chronic leukemia and suggested that interruption of pregnancy be carried out as early as possible since there was also exacerbation of the disease with later therapeutic abortions at term. Most authors agree with Sheehy's report[17] which showed statistically that the probability of survival was not impaired in patients with chronic leukemia and pregnancy. Some investigators[4, 54] even quote the beneficial effects of pregnancy on chronic leukemia.

Immediate exacerbation after delivery or abortion, especially in acute leukemia, can take place, although this may only show the short mean total duration of the disease (4 months) as well as its terminal complications. All 8 patients with acute leukemia and pregnancy died within 2 months after delivery or abortion in our series, with an average of 18 days.

It has been shown that treatment with the newer antileukemic drugs in addition to steroids, antibiotics and transfusions has improved maternal survival. In acute leukemia and pregnancy the improved survival appears to be chiefly due to steroids[2, 25, 55, 56, 57] and 6-mercaptopurine.[18, 25, 58] Levin et al[59] showed methyl guanyl hydrazone to produce remission in almost 1 out of 2 patients with acute myeloblastic leukemia. Corticosteroids are more beneficial in acute lymphoblastic leukemia, whereas in acute granulocytic leukemia few remissions result, with even severe myeloproliferative responses having been reported.[22, 60-69] The acceleration by corticosteroids was not observed frequently when they were used in conjunction with myelosuppressive drugs.[69] It has been recommended that neither be used during the first trimester. Since amenorrhea occurs not infrequently in leukemia, pregnancy should be ruled out before antimetabolite or radiomimetic therapy is instituted. If steroids need to be used early in pregnancy, they should be used only in small doses (25 to 50 mg. of prednisone per day),[18] because they pass the placenta,[70] and fetal abnormalities and adrenocorticoid insufficiency in the newborn have been reported in association with corticosteroid therapy in the mother.[71, 72] 6-Mercaptopurine is teratogenic in animals,[73, 74, 75] and a stillbirth has been recorded following its use for criminal abortion.[76]

In the series presented in this paper the results of the rational treatment outlined above seemed to have been beneficial to the mother and did not appear to harm the fetus. Thus, those pregnant patients who developed leukemia in the second or third trimester delivered viable infants in two-thirds of the cases when they were treated with steroids and 6-mercaptopurine. In acute leukemia fetal mortality was higher if the disease occurred early in pregnancy whether there were concentrated efforts at treatment or not. Whether the abortifacient factor is the effect from the disease itself or from the 6-mercaptopurine is difficult to determine. One study[25] based the response to therapy upon the morphologic distinction between lymphoblastic and myeloblastic leukemia and showed essentially the same results. That is, patients with lymphoblastic leukemia responded to steroid therapy and often to 6-mercaptopurine and antimetabolites of folic acid, whereas only few of those with myeloblastic leukemia showed improvement with 6-mercaptopurine and only rare remissions occurred with the other drugs.

Antifolic acid agents have produced fetal anomalies in individuals who had used the drug for attempts at criminal abortion.[77, 78, 79]

In table 2 it is shown that the treatment in chronic leukemia has greatly reduced maternal mortality. In the myelogenous type radiotherapy to the spleen and 1–4 dimethanesulfonyloxybutane (busulfan, Myleran®) consistently give the best results. In the lymphocytic type radiotherapy in combination with chlorambucil seems to be the method of choice.[55, 80] To support 1–4 dimethanesulfonyloxybutane (busulfan) in the former, many reports have shown that it aided the mother to deliver viable infants.[29, 31] Furthermore, there is documentation of 7 patients with chronic myelogenous leukemia who were treated with it during the first trimester.[81-87] There was only 1 in this group who reported congenital malformations in the fetus.[84] Some of the anomalies in this fetus included bilateral microphthalmia, bilateral corneal opacities, cleft palate, hypoplasia of the ovaries, stunting of growth (weight at term 1,077 Gm.), and cytomegaly. However, this patient had also received radiotherapy at approximately 2 weeks of gestation as well as 6-mercaptopurine. One mother in this series who delivered a stunted hypoplastic stillbirth had received 6-mercaptopurine and steroids.

Whether leukemia per se can be transmitted from mother to the fetus remains a controversial issue. There is no concrete evidence that transmission of leukemia from mother to fetus can take place. Even though the follow-up studies of the children born to leukemic mothers were not carried out in this investigation, others have shown no correlation. Diamandopoulos and Hertig[88] and Finch[89] reported no apparent evidence of transfer to the fetus. Bjure[90] did a follow-up study in children and grandchildren and found no evidence of transfer of chronic myeloid leukemia. However, Cramblett et al[91] reported a case of a 9-month-old baby who died of acute lymphatic leukemia after birth to a mother with acute leukemia.

It was shown that placental transmission took place in 3 of 32 cases of Hodgkin's disease.[92] This finding increased similar investigations in leukemia. Rigby and his colleagues[93] showed passage of quinacrine-labeled leukemic cells across the placenta. Histologic evidence from autopsy has also demonstrated placental transmission.[26] Bierman[62] discussed this problem in a comprehensive presentation. Bernard et al[39] demonstrated a possible trisomy 15 and trisomy 22 in an infant who developed leukemia 5 months after being born to a mother afflicted with acute leukemia. In another child, myelogenous leukemia developed at 3 years of age, but the mother had also received irradiation to the spleen at 16 weeks' gestation.[94] Even though leukemic cells have been shown to pass the placenta and though there have been infants developing leukemia after being born to leukemic mothers, this does not establish that communication of the disease has taken place. However, the correlation of such findings in only 1 or 2 cases in approximately 304 reported cases of leukemia during pregnancy probably places the incidence much higher than the 4 per 100,000 in this age group in the general population.[18]

Summary

Pregnancy complicated by leukemia has been reviewed, with the contribution of 9 cases. The observation that nearly 80 per cent of these patients had A-positive blood group was presented as a new finding. Leukemia in the pregnant patient was not considered more infrequent than in the nonpregnant female. The disease seems to occur more commonly in the Caucasian woman in her late 20's, and acute leukemia predominates over chronic. Exacerbation after delivery or abortion is common and fatal, especially in acute leukemia. Maternal mortality during pregnancy and parturi-

tion in chronic leukemia has been very low. Treatment with the newer antileukemic drugs* in addition to steroids, antibiotics and transfusions has improved maternal survival although some may have had teratogenic effects.

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* During the completion of this manuscript many new antileukemic drugs have appeared for clinical investigational trials, and some results have been encouraging. Their use in the pregnant patient has been limited because of the drugs' unknown effects upon the fetus, although one should consult the literature for the current status of each such drug.

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(*Continued on page 632*)

Clinical and Hemodynamic Features of Idiopathic Hypertrophic Subaortic Stenosis (IHSS)

Report of Four Cases[*]

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THE SYNDROME OF IHSS, which is an obstructive myopathy of the left ventricular outflow tract, has received considerable attention over the past 10 years, and many excellent publications have detailed its various clinical and hemodynamic features.[1-6] Nevertheless, in many cases it continues to go unrecognized for several years. For this reason and because of the great interest in its hemodynamic features presentation of the following 4 cases is considered justified.

Report of Cases

Case 1. C. B., a 39-year-old Negro alcoholic woman, was found in 1961 to have high blood pressure and a heart murmur diagnosed as rheumatic mitral regurgitation or aortic stenosis. In late March 1968 she began to complain of fatigue, effort angina and dyspnea, and paroxysmal nocturnal dyspnea. She had dizziness and 2 syncopal episodes during the first 3 months of 1968. The findings of a bifid arterial pulse, bifid apical impulse, and a loud, harsh ejection systolic murmur along the left sternal border prompted admission for diagnosis on April 3, 1968. Personal and family histories were unremarkable. The patient was slightly obese but otherwise in apparently good health. The jugular venous pulse (JVP) was normal but arterial pulses were rapidly rising with a distinctly bifid character (figures 1 and 2). The apical impulse was displaced to the left and was of a diffuse, strong and bifid quality without presystolic atrial "kick" (figure 1). A systolic thrill was felt along the lower left sternal border. Heart sounds were physiologic, and there were no gallops or diastolic murmurs. A harsh ejection systolic murmur, grade 4/6 was maximal in the fourth space to the left of the sternum and radiated widely both to the left axilla and to the neck (figure 1). The electrocardiogram (ECG) showed a QRS axis of −10°, left ventricular hypertrophy, and ischemia. A relatively wide Q wave was present in lead aVL (figure 3). The chest roentgenogram showed left ventricular (LV) enlargement.

Hemodynamic data. Noteworthy features included a prominent left atrial "a" wave with a corresponding "kick" in the LV pressure pulse, slightly elevated left atrial (LA) mean pressure, right ventricular (RV) systolic and diastolic pressures, and pulmonary artery (PA) mean pressure. With a transseptal catheter lying in the left ventricular inflow (LVI) and a retrograde aoritc catheter placed in the LV outflow (LVO), it was possible to demonstrate a clear hemodynamic separation of the LV cavity into a high-pressure inflow and low-pressure outflow region. As noted in tables 1 and 2 and figures 2, 4 and 5, the high LVI-LVO systolic gradient became even higher after intravenous infusion of isoproterenol at a rate of 4 mcg./min., after premature ventricular beats (PVB) and after inhalation of amyl nitrite. The same observation was made during the Valsalva maneuver. A smaller gradient appeared between LVO and brachial artery after all these maneuvers. An LV angiogram showed a small LV cavity surrounded by exuberant myocardial masses (figure 6). This patient has been followed in the Clinic since this study and has developed mild LV failure. Surgery has been contemplated.

Comment. In this case the murmur was mislabeled for several years and its true nature was recognized only when due attention was given to the characteristics of the arterial pulse and the apical impulse.

Case 2. B. O., a 20-year-old Caucasian woman, was referred to our laboratory for confirmation of the diagnosis of IHSS entertained by the referring physician.[||] A heart murmur discovered at the age of 6 years could not be attributed to rheumatic fever or other known causes. She remained asymptomatic until the age of 17 when she developed a progressive dyspnea on exertion culminating in severe orthopnea and paroxysmal nocturnal dyspnea. She became totally incapacitated and had a therapeutic abortion 3 months before admission, following which she was in shock for 3 days. Digitalization, begun 6 months before admission, failed to improve symptoms. She had had 2 syncopal attacks with moderate exertion. The family history was negative with respect to heart disease, sudden death, and diabetes. She was slightly obese and appeared to be in excellent general health. The blood pressure was 110/70 and had never been known to be elevated. The patient became moderately dyspneic on walking. The JVP was normal but the peripheral arterial pulses were hyperdynamic and rapidly rising (figure 7). The apical impulse was diffuse and hyperdynamic with 3 components judged to represent an atrial "kick" and a bifid ventricular systolic impulse.

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A harsh, grade 4/6, long ejection-type murmur was maximal at the apex and audible over a wide area around the apex and along the left sternal border, not extending significantly to the right of the sternum or the neck. A loud atrial gallop was heard maximally at the apex but was audible over the entire left anterior chest (figure 7). Murmurs of mitral and aortic regurgitation were not heard. The QRS axis was at −10°. There was evidence of left atrial enlargement, left ventricular hypertrophy, and ST-T changes of LV ischemia and possibly additional digitalis effect (figure 3). The chest roentgenogram showed LV enlargement.

Hemodynamic data. The significant findings were a prominent atrial systolic wave transmitted to the LV apex, LV pressure pulse (figure 8) and the peripheral arteries (figure 7), and a modest pressure gradient between LVI and LVO with magnification after pre-

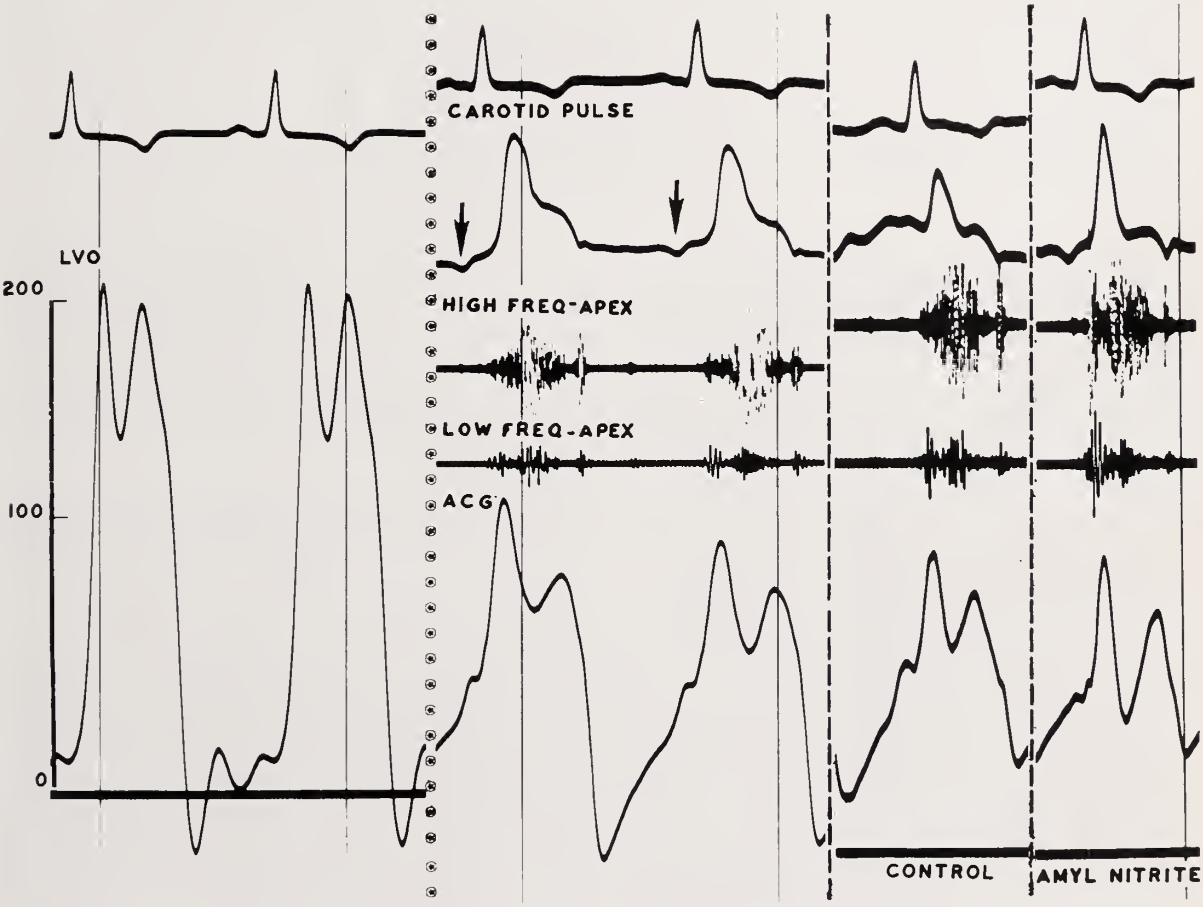

FIG. 1. Case 1, C. B. Left hand panel is pressure pulse recorded from LV outflow and shows the characteristic midsystolic trough corresponding to midsystolic trough of apical impulse as depicted in right-hand panels. Note bifid impulse recorded on apex cardiogram (ACG) and its magnification after inhalation of amyl nitrite. Corresponding to LV midsystolic trough, peripheral arterial pulses show a dip which imparts to them the characteristic bisferious or bifid configuration. The left ventricular outflow is not obstructed in early stages of ejection, and therefore first parts of LV pressure pulse, ACG, and arterial pulse proceed normally or even more vigorously than normal. However, progress of ejection is interrupted in midsystole by contraction of hypertrophied basal septum causing obstruction. Panels on right depict negative atrial imprint (arrows) and modest midsystolic trough in carotid pulse tracing. Diamond-shaped murmur recorded from apex and apex cardiogram with its midsystolic dip are also shown.

TABLE 1: Symptoms and Signs in 4 Cases of Idiopathic Subaortic Stenosis

Patients	Dyspnea			Chest pain	Palpitation	Syncope	S.D. in family	HBP	LVH	Apex Impulse	ADG	Thrill	SM	DM	JVP	BA	CHF
	Orthopnea	DOE	PND														
C. B.	−	+	+	+	−	+	−	+	+	bifid	−	+	+	−	N	bifid	+
B. O.	+	+	+	−	−	+	−	−	+	bifid	+	−	+	−	N	bifid	+
D. K.	+	+	−	−	+	−	−	+	+	bifid	+	−	+	−	N	bifid	+
R. W.	?	?	?	?	?	+	?	−	+	bifid	+	+	+	+	N	N	−

DOE: Dyspnea on exertion
PND: Paroxysmal nocturnal dyspnea
S.D.: Sudden death
HBP: High blood pressure
LVH: Left ventricular hypertrophy
ADG: Atrial diastolic gallop
SM: Systolic murmur
DM: Diastolic murmur
JVP: Jugular venous pulse
N: Normal
BA: Brachial artery
CHF: Congestive heart failure

mature ventricular beats and following long pauses brought about by occasional 2° AV block which occurred during the procedure (tables 1 and 2 and figure 8).

After the clinical impression of IHSS was verified by these studies, the patient was advised to stop taking digitalis. This was followed by considerable improvement in all her symptoms to the point that she was able to resume full activity and conceive and deliver a normal child. Two years later the symptoms recurred and necessitated surgical relief of obstruction with excellent results.* At present she continues to enjoy excellent health.

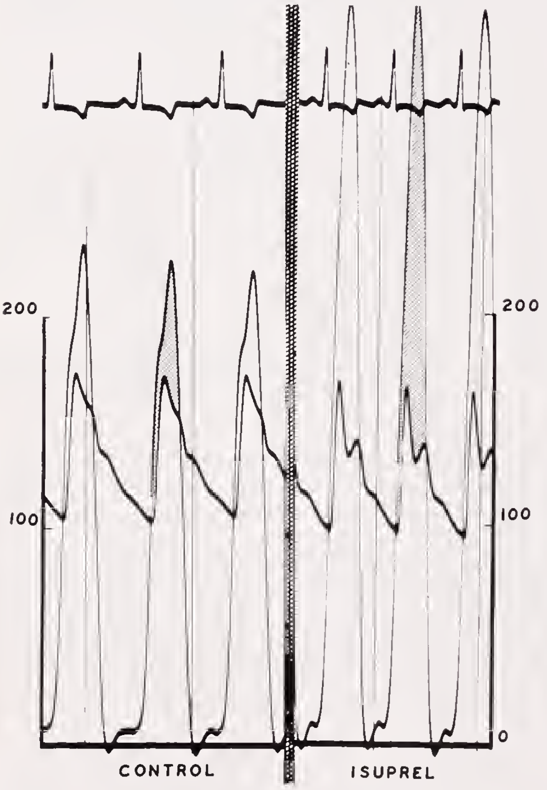

FIG. 2. Case 1, C. B. LV inflow and brachial arterial (BA) pressures recorded at the same amplification before and after intravenous infusion of isoproterenol, 4 mcg./min. Note marked exaggeration of LV-BA gradient, magnification of the midsystolic trough in the BA tracing, and the peaking of arterial pressure in early systole in contrast to late peaking in LV pressure.[7]

Comment. This was a typical case of IHSS, recognizable clinically and documented easily by hemodynamic investigations. The unfavorable response to digitalis and the excellent surgical outcome are noteworthy.

Case 3. D. K., a 69-year-old Negro man, had a known heart murmur and high blood pressure for a number of years. His admission for diagnostic studies in September 1964 was prompted by the development of palpitation, orthopnea, and exertional dyspnea. IHSS had been suggested by the brisk peripheral pulses and the harsh ejection murmur and thrill to the left of the sternum. He had had no fainting or dizziness or anginal pains. Personal and family histories were negative. The arterial blood pressure was 170/110, and the pulse was of a brisk, rapidly rising quality; the JVP was normal. The apical impulse was bifid and displaced leftward and inferiorly. A coarse thrill was palpable along the lower left sternal border. A harsh, grade 5/6, long ejection systolic murmur was maximal at the apex and lower left sternal border and radiated to the base but not to the neck. No atrial or ventricular gallops could be heard, but an atrial sound was recorded on the phonocardiogram (figure 9). The ECG showed bi-atrial enlargement but no LV hypertrophy (figure 3). The chest roentgenogram suggested LV enlargement.

Hemodynamic data. There were a modest RV hypertension, raised left atrial "a" wave, and a mild LV-AO systolic gradient with no post PVC magnification

---

* Surgery was performed by Dr. D. A. Cooley, Saint Luke's Episcopal Hospital, Houston, Texas.

TABLE 2: Hemodynamic Data

Patients	RA			RV	PA		LA			Basal State				Post PVB			Isoproterenol			Amyl Nitrite			Atropine		
	a	v	m	s/d	s/d	m	a	v	m	LVI	LVO	Aorta	Gr	LVI	LVO	Gr	LVI	LVO	Gr	LVI	LVO	Gr	LVI	LVO	Gr
C. B.	8	5	4	35/8	37/19	28	18	24	17	240/7	141/7		99*	345	135	210	314	143	171	238	100	138			
B. O.				20/−5 to 0	20/12				5–10	164/5–20		110/78	54	210	130	80									
D. K.	7	2	5	44/5	42/20	22	17	12	12	144/10		110/60	34	128	94	34	158	98	60						
R. W.	8	5	6		20/10	13	12	12	7	155/14		155/90	0	190	110	80	210	100	110	152	111	41	164	146	18

RA: Right atrium
RV: Right ventricle
PA: Pulmonary artery
LA: Left atrium
m: Mean pressure

s/d: Systolic/diastolic
LVI: Left ventricular inflow
LVO: Left ventricular outflow
Gr: Peak systolic pressure gradient
PVB: Premature ventricular beat

* Pressure in the aorta was generally at the same level as in LVO, and therefore LVI-aorta gradients which were the same as those of LVI-LVO are not recorded separately.

(tables 1 and 2). The gradient became greater after I.V. infusion of isoproterenol. This patient has been treated medically and continues to do well with restricted activities. Surgery has been refused.

Comment. The advanced age of this patient with IHSS is particularly noteworthy.

Case 4. R. W., a 68-year-old Negro man, was admitted to the Psychiatric Division of this hospital because of what was diagnosed as chronic brain syndrome. He had had 3 episodes of fainting and convulsion during the 3 months before admission but complained of no cardiac symptoms. His mental confusion precluded a reliable history. He was normotensive and exhibited no evidence of heart failure. The peripheral arterial pulses and the apical impulse lacked the features of IHSS. There was a systolic thrill along the upper right sternal border with a harsh, grade 3/6 ejection-type systolic murmur in the same area. The murmur was easily heard at the apex. A grade 2/6 diastolic murmur of aortic regurgitation was heard

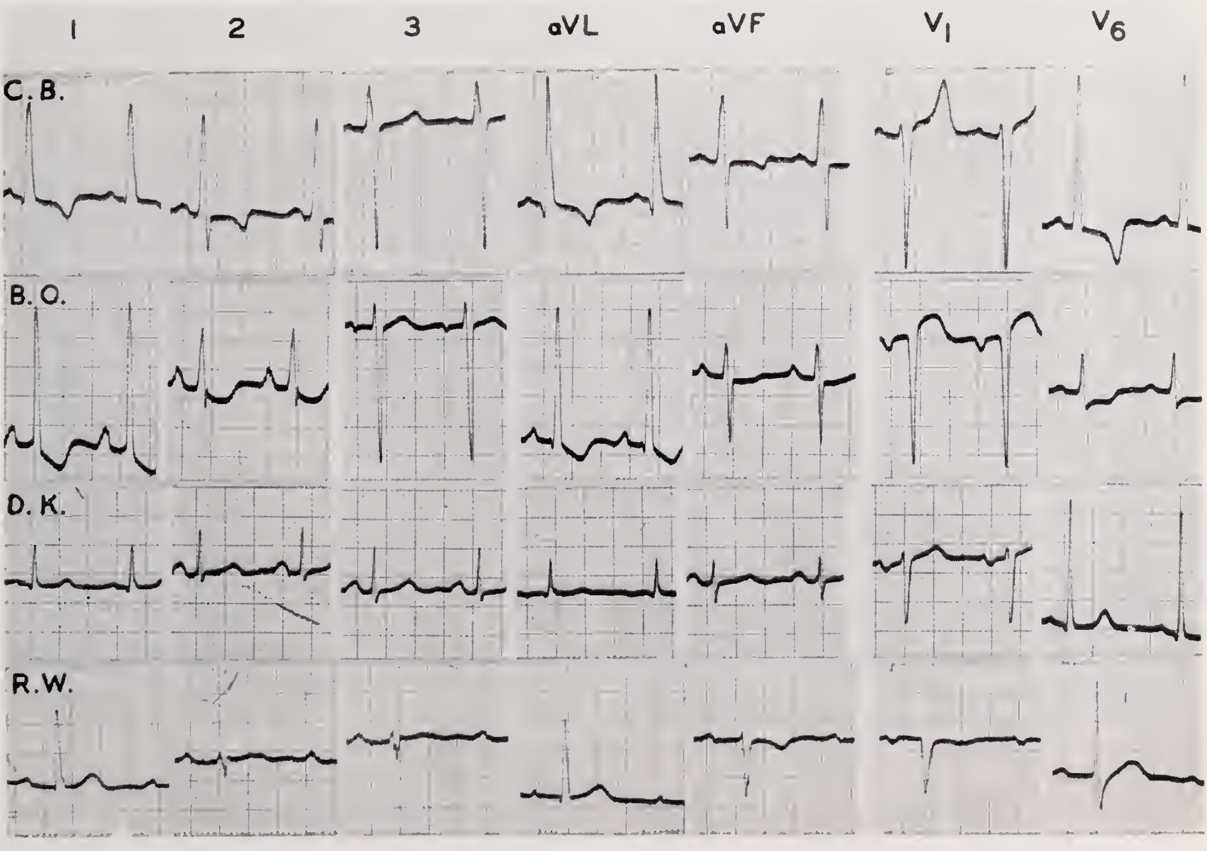

FIG. 3. Electrocardiograms in all 4 cases.

along the left sternal border. There was an atrial gallop at the apex. The ECG showed first degree AV block and suggested LA and LV enlargement (figure 3). The chest roentgenogram displayed slight LV enlargement.

Hemodynamic data. These were surprising in that no systolic gradient was found between the LVI and the brachial artery at rest. However, I.V. infusion of isoproterenol, inhalation of amyl nitrite, and I.V. injection of 1 mg. of atropine sulfate all gave rise to a systolic gradient (tables 1 and 2 and figure 10). Retrograde approach to the LV cavity through the femoral artery was unsuccessful because of extreme tortuosity of the iliac vessels. It should be emphasized therefore that the presence of a LVI-LVO systolic gradient such as that shown in case 1 was not demonstrated in this patient and, further, that cavity obliteration with resultant catheter entrapment and the spuriously high LV pressure[8] could not be ruled out.

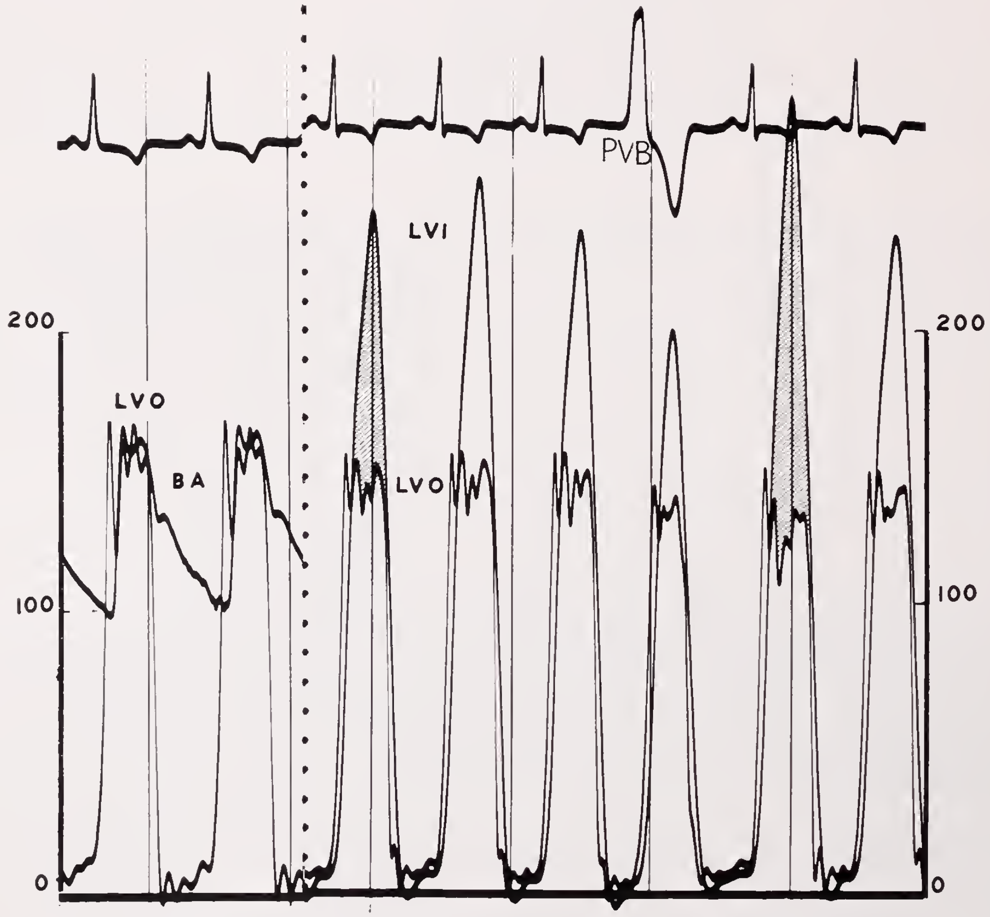

FIG. 4. Case 1, C. B. Simultaneous LV outflow and BA pressures in left-hand panel show no gradient. Simultaneous LV inflow and outflow pressures in right-hand panel document existence of a gradient between these 2 regions. Note post-PVB magnification of gradient.

Comment. In this case with clinical features of valvular aortic stenosis, hemodynamic data favored IHSS. However, our inability to advance the catheter through the iliac arteries precluded the demonstration of an intracavitary LV gradient. The presence of a diastolic murmur is of particular interest.

Discussion

The syndrome of IHSS is now accepted as a form of cardiomyopathy in which there is a disproportionate hypertrophy of the interventricular (IV) septum and particularly its upper zone just below the membranous region. The bulging IV septal mass, together with distorted anterior leaflet of the mitral valve, is presumed to be the cause of obstruction in the outflow region of the left ventricle.[9] The magnitude of obstruction is necessarily variable because it depends on the vigor of myocardial contraction and the volume of the LV cavity. The presence of this obstructing muscle mass creates a pressure gradient within the cavity of the left ventricle between its inflow tract and outflow tract immediately below the

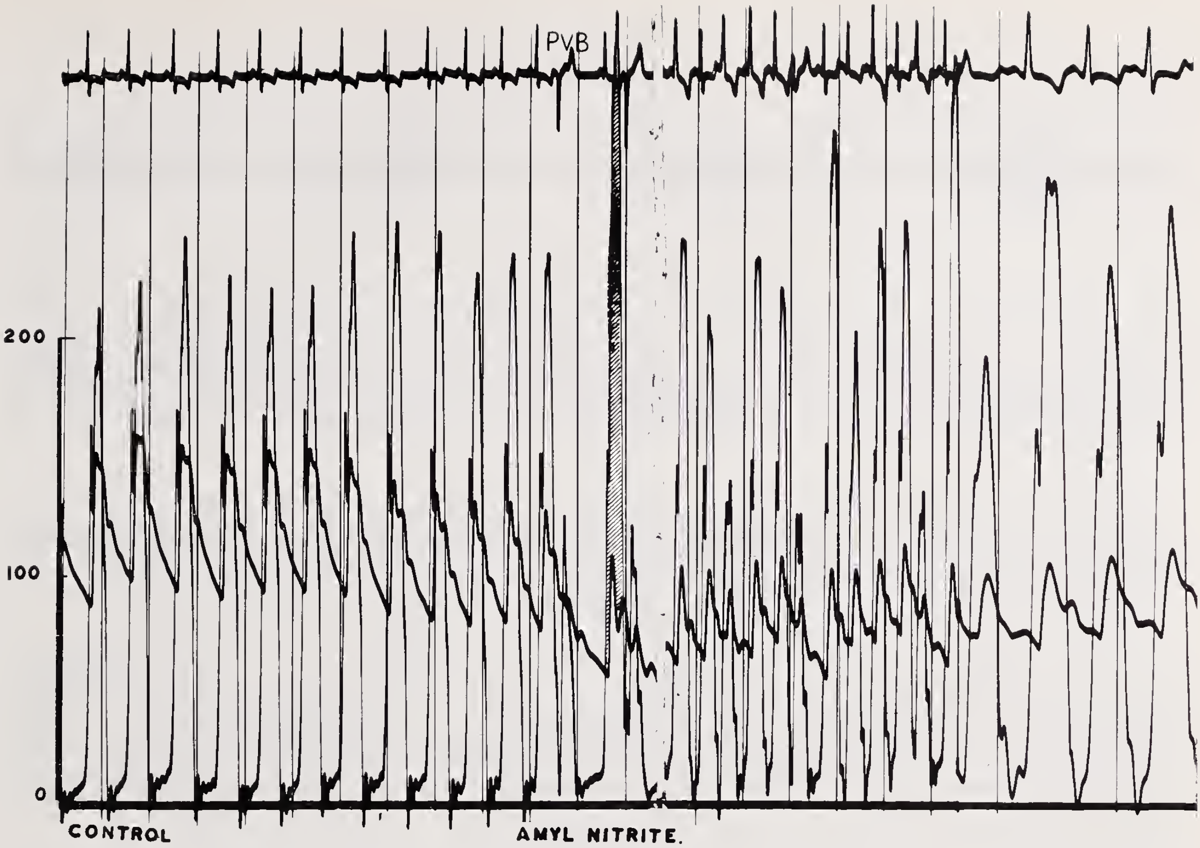

FIG. 5. Case 1, C. B. LV inflow and BA pressures recorded at equal amplifications. First 6 beats were recorded before effect of amyl nitrite became apparent. Note acceleration of heart rate, fall in arterial pressure, and increase in LVI-BA gradient. The post-PVB magnification of gradient became most striking after amyl nitrite as seen in middle of tracing. Toward right-hand extremity of tracing, records were made at a faster speed in order to show characteristic contour of LV pressure curve and midsystolic trough in BA pressure pulse.

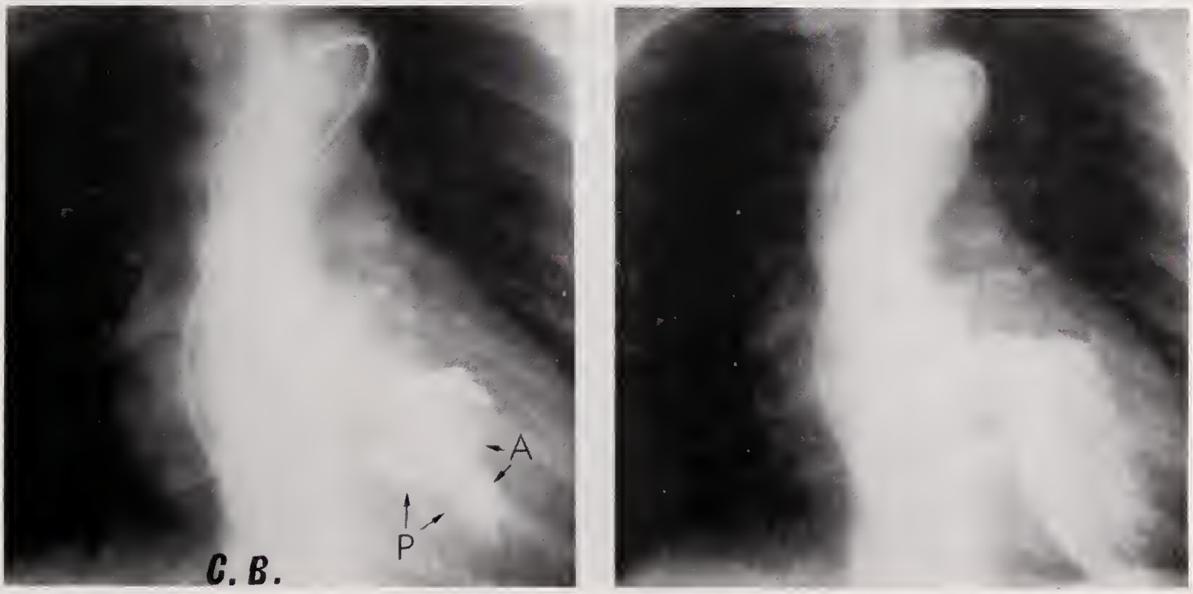

FIG. 6. Case 1, C. B. Left ventricular angiogram obtained during systole on left and during diastole on right. Note small systolic volume of left ventricle and bulges produced by the enormously hypertrophied anterior and posterior papillary muscles (A and P). Filling defect visible just below aortic valve is caused by hypertrophy of interventricular septum which is most pronounced in its basal region.

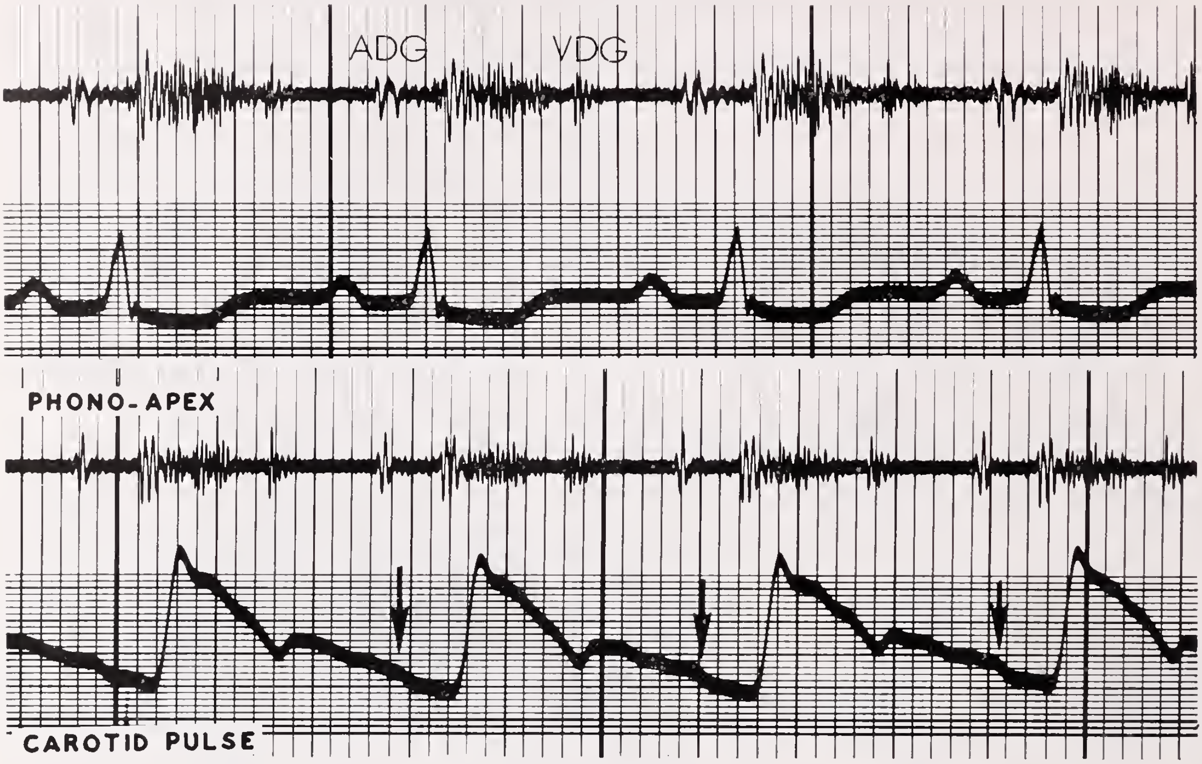

FIG. 7. Case 2, B. O. Phonocardiograms recorded from apex simultaneously with electrocardiogram (above) and carotid pulse (below). Note very loud atrial gallop, long ejection murmur, diastolic gallop, and atrial imprint on carotid pulse (arrows).

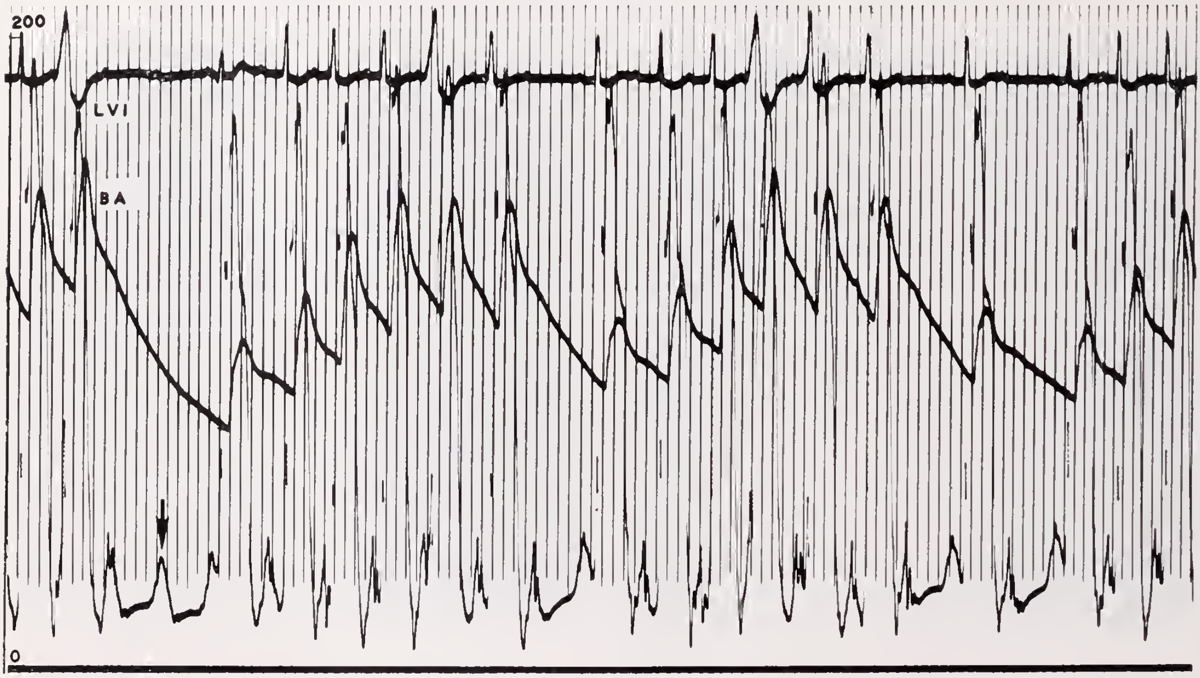

FIG. 8. Case 2, B. O. Left ventricular inflow and BA pressures recorded at equal amplifications after patient had developed second degree AV block during the procedure. Note that arterial pulse pressure is small and LV pressure unchanged after the long pauses. Result is a magnified pressure gradient. Note also strikingly forceful wave transmitted to ventricle at time of atrial systole (arrow).

aortic valve. There may be no or little gradient between the outflow tract and the ascending aorta as was demonstrated in our case 1. The possibility that the pressure gradient may be altogether artifactual and caused by entrapment of the catheter tip within the vigorously contracting trabeculae carneae of the apical region was elegantly demonstrated by Criley and co-workers.[8] However, the surgical experience with relief of obstruction after excision of the hypertrophied IV septum[3, 10] and the postmortem observations[3] leave little doubt that true obstruction does exist in many cases.

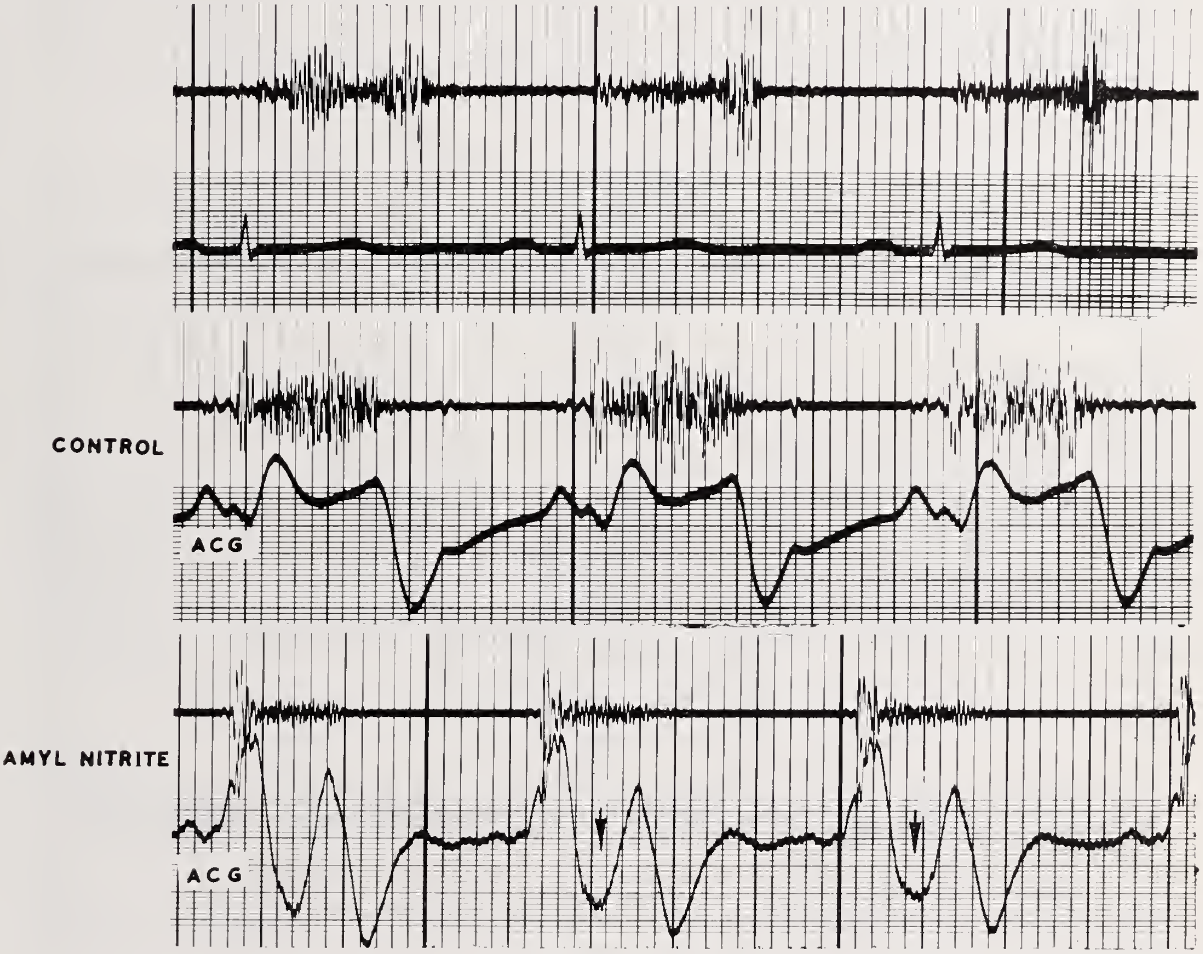

FIG. 9. Case 3, D. K. Phonocardiogram recorded in fourth left space near sternal edge (top), at apex simultaneous with apex cardiogram (middle), and after inhalation of amyl nitrite (bottom). Note atrial gallop and atrial "kick" recorded at apex (middle strip); also note pronounced midsystolic trough in ACG after amyl nitrite inhalation (arrows).

The etiology of the syndrome is unknown. A significant proportion of the cases follows the pattern of familial transmission. Other cases occur sporadically and may represent unusual forms of cardiomyopathy or the advanced stages of hyperkinetic heart syndrome.[3] The syndrome usually occurs in middle-aged persons.[6] Our experience with 2 patients in their sixties is worth noting and indicates the need to consider this diagnosis irrespective of the age.

The clinical symptoms are quite similar to those of valvular aortic stenosis (AS). Thus, dyspnea on exertion, anginal pain, syncopal attacks, and sudden death are the outstanding features of both types of LV outflow obstruction. Asymptomatic cases exist in both varieties. The distinct clinical improvement in 6 of the 28 patients with IHSS initially in Class III and IV of N. Y. Heart Association Classification[6] may be considered an important differential feature between this and the valvular AS. Certainly, the familial occurrence of the syndrome in various stages in one-third of the cases of IHSS[6] is also at variance with the findings in valvular AS and points to the need for careful examination and even hemodynamic study of the members of the family whenever this diagnosis is entertained.

Physical findings in IHSS, on the other hand, are quite diagnostic and often sufficient to differentiate it from valvular AS. It must be emphasized that certain cases may exhibit features of both diseases as was seen in case 4 of the present report. One wonders if the association of outflow muscular hypertrophy secondary to valvular stenosis of the type described by Brock[11] may not be responsible for such findings. Other conditions capable of masquerading as IHSS include mitral regurgitation and the hyperkinetic heart syndrome. It is to be pointed out, however, that mitral regurgitation may be a concomitant of IHSS by virtue of the distortion of the mitral leaflets.[9]

The features of IHSS which arouse suspicion include the quality of the peripheral arterial pulses, the character of the apical impulse, the characteristics of the systolic murmur, and the atrial gallop. All these features are magnified by the so-called provocative maneuvers designed to aggravate the obstruction through increasing LV contractility (isoproterenol,[3] digitalis,[3] exercise,[3] and atropine[12]), decreasing the LV volume thereby approximating LV walls (Valsalva maneuver,[13] amyl nitrite), and increase in LV contractility which occurs at the end of the long pause following premature ventricular beats, the so-called post-PVB phenomenon.[1] It has been shown in this study that the long cycle leading to a magnified pressure gradient need not be postextrasystolic and may be caused by 2° AV block (figure 8). Furthermore, the beat occurring at the end of the long cycle need not be a normal sinus beat and could be a ventricular escape (figure 11).

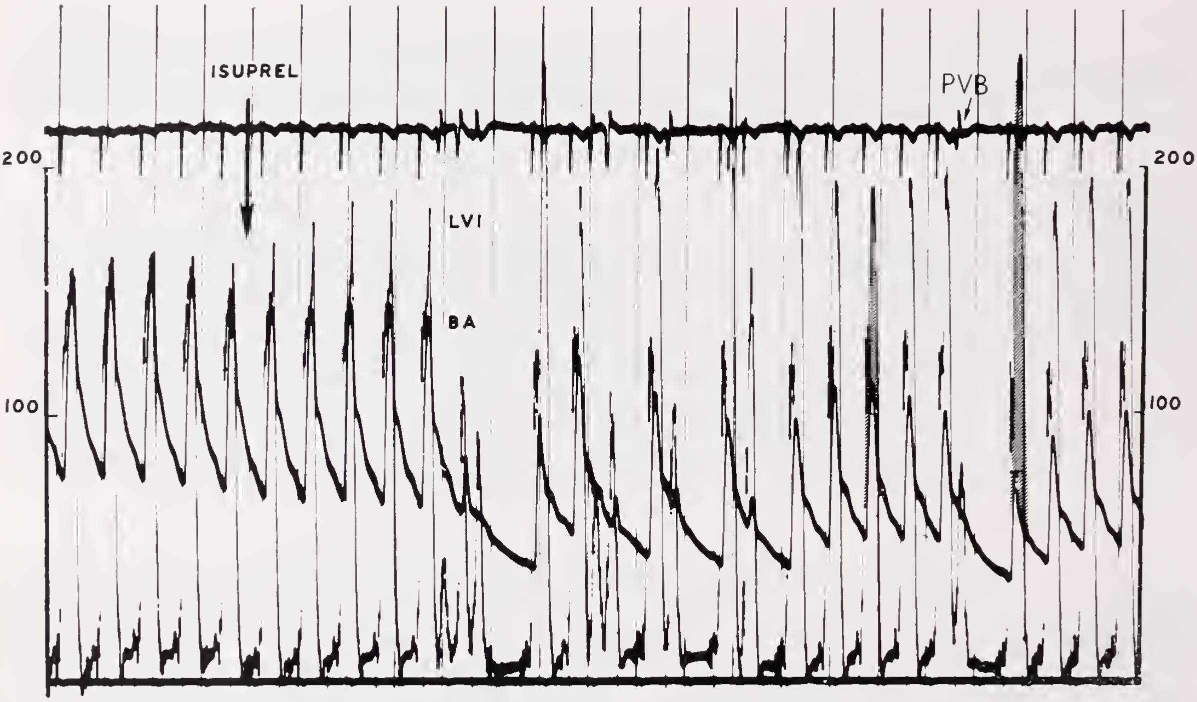

FIG. 10. Case 4, R. W. Simultaneous LV inflow and BA pressures at equal amplifications. First 5 beats were recorded just before intravenously infused isoproterenol exhibited its effect. Note absence of systolic gradient in control beats and gradually increasing gradient after isoproterenol. Increase in gradient is accounted for both by a fall in BA pressure and a rise in LV pressure which becomes particularly pronounced after the PVB.

The peripheral arterial pulse is characterized by a rapid rise and a mid-systolic trough. The former is reminiscent of the pulse in mitral regurgitation, and both features are similar to those in the hyperkinetic heart syndrome. Our cases have brought to light a rare feature, a diminished LV compliance and forceful atrial contraction, with an imprint on the arterial pulse tracing by the left atrial contraction (figures 1 and 7). This imprint, which was positive in 3 cases and negative in 1, was not detected by the examining finger. Its genesis is obscure and most likely represents forceful contraction of the left atrium imparting ballistic forces to the left ventricle and the aortic root. Review of the published tracings of IHSS reveals this feature in some cases. We have made similar observations in patients with primary myocardial disease.[14] The bifid quality of the pulse may be negligible or absent before the provocative maneuvers (figure 2). The midsystolic dip of the apical impulse is particularly helpful when it is magnified by the provocative maneuvers and if it is associated with a palpable atrial "kick" at the apex (figures 1 and 9). The systolic murmur, which is an invariable finding, is usually harsh, diamond-shaped, and loudest either along the left sternal border, at the apex, or both. It is often associated with a thrill. Its location is the most helpful differential feature as compared with valvular AS.

The presence of a diastolic murmur of aortic regurgitation need not mitigate against the diagnosis of IHSS. In one large series of 126 cases 7 patients had diastolic murmurs,[6] as did our case 4. The atrial gallop which is often both palpable and audible at the apex is not peculiar to this syndrome and is commonly found in valvular AS, acute mitral regurgitation, hypertension, and the hyperkinetic heart syndrome. In our series atrial gallop was present in 3 cases and absent in 1. This compares with the finding of atrial gallop in 56 of 63 patients by Braunwald et al.[3]

The electrocardiogram shows left ventricular hypertrophy in 70 per cent of the cases, and very large septal Q waves suggesting myocardial infarction have also been found.[6] In the present study the latter feature was lacking, but LV hypertrophy was present in 3 cases. The chest roentgenogram usually shows LV enlargement.

The hemodynamic documentation of IHSS requires left-heart catheterization and not infrequently the use of the provocative tests as used in the cases reported here. Catheter entrapment as a cause of high intraventricular pressure must be ruled out by demonstrating a gradient during pullback across the outflow tract, or, preferably, by recording pressures through 2 catheters, one

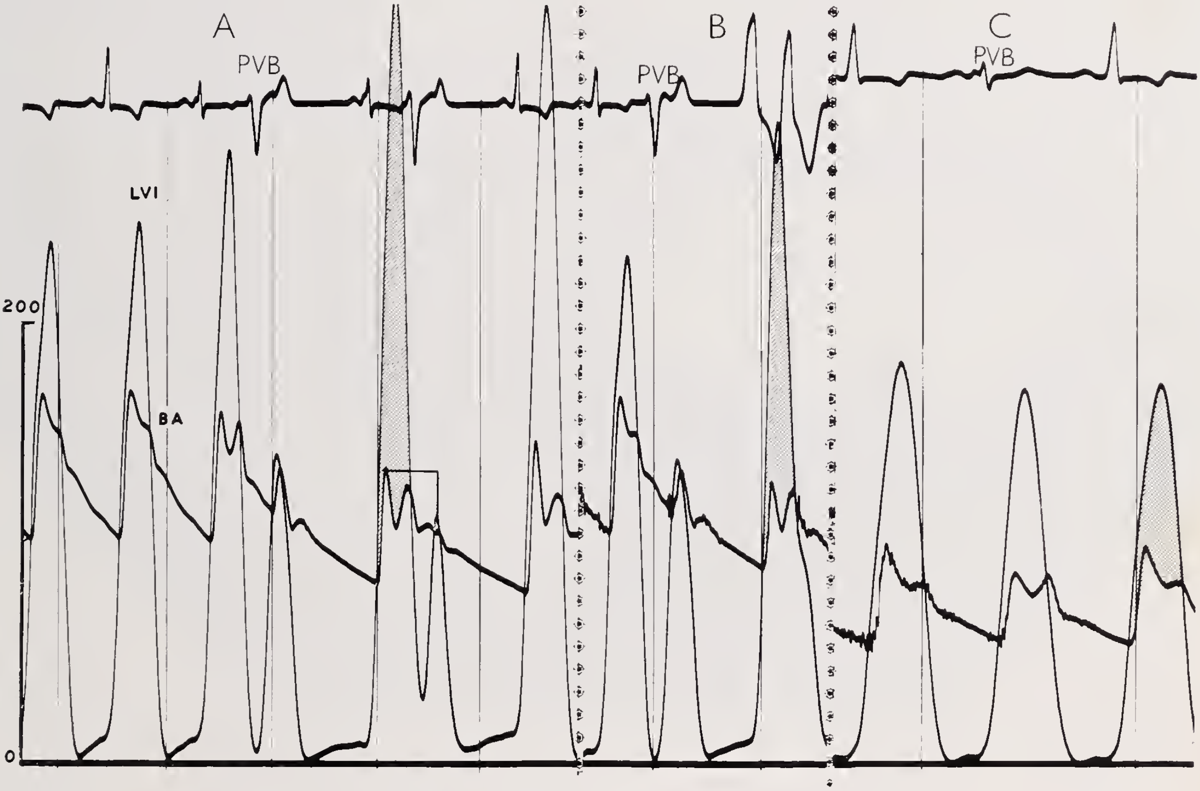

FIG. 11. Case 1, C. B. Simultaneous LV inflow and BA pressures recorded with 3 different types of arrhythmia for purpose of demonstrating importance of length of cardiac cycle in determining magnitude of pressure gradient irrespective of electrocardiographic configuration of beat occurring at end of long cycle. In panel A the magnified gradient, which is shaded, appears after a post-PVB pause even though QRS configuration of post-PVB beat is not normal. In panel B the beat occurring after the PVB is actually a ventricular escape. Nevertheless, gradient for that beat is magnified. In panel C the PVB is very late and occurs almost simultaneously with a normally conducted beat (fusion). Consequently it is not followed by a pause and the post-PVB gradient is not magnified (shaded area).

of which lies in the inflow and the other in the outflow tract of the LV. Ancillary hemodynamic findings in IHSS include a large "a" wave in both atria, mild pulmonary hypertension, and occasional gradient across the right ventricular outflow.

Left ventricular angiography shows 3 cardinal abnormalities consisting of a small LV cavity in systole, filling defects brought about by the massively hypertrophied papillary muscles and the upper part of the IV septum best seen in the lateral view, and mitral regurgitation (figure 6). For further details of the hemodynamic and angiographic parameters the reader is referred to the excellent review articles on this subject.[2, 3, 6, 9]

Treatment includes measures designed to decrease myocardial contractility such as the administration of beta adrenergic blocking agents[15] and surgical excision of the hypertrophied portions of the IV septum.[9, 16, 17]

Summary

Four cases of IHSS have been presented and the useful clinical clues have been pointed out. It has been indicated that hemodynamic documentation by means of left-heart catheterization and left ventricular angiography is often necessary, and the appropriate technique has been described.

It is suggested that careful attention to the quality of the peripheral arterial pulses and the apical impulse in patients with systolic ejection murmurs will enable the examiner to suspect the correct diagnosis and proceed with hemodynamic and angiographic documentation.

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Grinszpan, Goldstein and Divito[2] used Lomotil in 20 children with diarrhea and also reported a prompt disappearance of diarrhea. Harris and Beveridge[3] in a double-blind study of 50 children with diarrhea, however, found no clear pattern to suggest that Lomotil influenced the course of the condition.

Michener, Brown and Turnbull[4] added evidence supporting the beneficial effects of Lomotil in 80 children, concluding that Lomotil was highly useful in controlling abdominal cramping, diarrhea and hypermotility.

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or 2 tablets q.i.d.

*Based on 4 cc. per teaspoonful.

Maintenance dosage may be as low as one-fourth the initial daily dosage.

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President's Page

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In this explosive age of scientific discovery no physician can keep abreast of progress without frequent, thorough reading of the medical literature. Students are questioned on the latest periodicals and are led, through journal clubs, into the habit of daily reading. Yet, strangely, little emphasis is placed upon the importance of acquiring the habit of medical writing. Hippocrates' Oath that "I will teach this art. . . by precept, lecture, and every other mode of instruction" seems to have been forgotten in the day-by-day trials of practicing physicians.

Not more than 1 doctor in 50 during his lifetime makes significant contributions to the world's vast storehouse of medical knowledge. The usual excuses are: "I can't write," "I am too busy taking care of my patients," or "There are too many worthless articles in the journals without my adding to them."

Doctors of Medicine are all college graduates and are perfectly capable of writing articles suitable for publication. If not, they can easily secure the assistance of a trained expert. If we are too busy to write, John Hunter, who slept only 4 hours a night and whose many writings on such subjects as the collateral circulation, aneurysms, and the Hunterian chancre established him as one of the 3 greatest surgeons of all time, was not. If we are too occupied in saving lives to write, how many million more lives were saved by Edward Jenner in his simple 1798 description of cowpox inoculation against smallpox than are saved by any one practitioner in his entire practice? How many million more lives were saved by Semmelweis in his 1846 analysis of the cause and prevention of puerperal sepsis than are saved by any one obstetrician? Certainly any busy practitioner encounters many perplexing problems and achieves many successful results which, if known by others, would prove to be invaluable. The choice of material for publication is determined by editors. The contributions must be made by individuals.

Medical writing is a matter of habit, and like all habits is best begun early in life. John Shaw Billings, the great educator, suggested: "There is nothing really difficult if you only begin; some people contemplate a task until it looms so big it seems impossible. I just begin and it gets done somehow. There would be no Coral Islands if the first bugs sat down and began to wonder how the job was to be done."

Those who profit by the world's scientific literature have an obligation to contribute to it.

William S. McCune

Editorials

Thanksgiving*

THANKSGIVING, incidentally, falling usually on the next to the last Thursday in November—long after the harvest has been gathered—comes at about the middle of the Christmas season, which has been stretched almost to the breaking point. Its first tinsel now appears soon after 12 October, celebrated hereabouts as the birthday of Christopher Columbus, credited with having discovered America. Whether this should be considered as a cause for thanksgiving is a subject for debate in some parts of the world, including the Indian reservations.

I admit to a personal approval of the idea of having at least one day in the year set aside for thanksgiving, especially since I have enough personal reasons for gratitude to require about 365 days for their full enjoyment. They are extraordinarily diverse—a retreat in the woods in which to meditate beside a wood fire; an indulgently Puritanical upbringing; opportunities to work, including many years in paediatric practice, followed by another score as an editor; a fair share of friends with some very special ones located between Deal and Ramsgate and the River Tweed; a family of which to be proud, and domestic felicity.

JOSEPH GARLAND, M.D.
Editor Emeritus, New England Journal of Medicine

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The George Washington University Design for Medical Education

FOR MORE THAN A CENTURY The George Washington University has prepared physicians and medical scientists for careers at home and abroad. Current social, economic and political conditions are severely testing the purpose and products of all universities, but there is little evidence that academic principles should be changed. Teaching programs, however, do require extensive refurbishing to fill the cultural and scientific needs of our Nation.

To meet the expanding dimensions in health care, the School of Medicine accepted the challenge of changing its curriculum from that of a fixed professional course to a university type of learning process. In 1962 the academic program was flexibly structured to meet the requirements of individual students through a balanced series of required and elective experiences.

The educational policy was progressively implemented with curricular reform in the clinical years, followed by changes in the preclinical years. The Class of 1967 was the first to benefit by the total 4-year program. Elimination of nonessentials and inclusion of new essentials required intelligent intradepartmental evaluation. The effectiveness of the new program was demonstrated this year when The George Washington University School of Medicine was the only medical school in the United States or Canada in which there were no failures among the second-year students who took the National Board Part I Examination in June 1968.

In our design for medical education, first- and second-year students have a strong program of elective courses in the basic medical sciences, including research opportunities. Each clinical department assigns students for inpatient experiences wherever the best hospital program among the 7 affiliated hospitals can be devised. Clerkship schedules have been revised with individual flexibility in research and career electives.

The new curricular revision has been tested. Now, plans are being made to wrap properly designed buildings around our educational concept. A detailed study of the physical resources needed to support the academic design indicated the advisability of providing new facilities nearer the University Hospital. The Medical Center

Opinions expressed in contributions to the Editorial Section are those of the writers and do not necessarily reflect the views of the Medical Society of the District of Columbia

is being expanded on a 4-block University campus site on Washington Circle.

Major new facilities will include a Medical Library, Basic and Clinical Sciences Buildings, and the University Clinic. While resources are being developed Medical Center teaching, research, and practice continue. The University Hospital has been enlarged and renovated to include a floor of research, a new surgical suite, X-ray therapy, and pharmacy sections. An expanded intensive care unit opened in September. On the Pennsylvania Avenue side of the Hospital an expanded emergency and trauma unit, heart station, and pulmonary and rehabilitation sections are now under construction. The Keystone Building, across 22nd Street, will serve as a pilot project for the new Clinic. The 12-story former apartment house has been redesigned to encompass an educational group practice in all specialties.

The Keystone experience will be applied in constructing the University Clinic. The Clinical Sciences Building will incorporate the latest electronic teaching devices in its lecture halls and amphitheaters. The Harry F. Duncan Cardiovascular Research Building, coördinated with the space of the present Warwick Building, will complete a research center in heart disease, cancer, and stroke.

The University Medical Center has designed a concept to be carried out by capable faculty members with qualified students in specially planned facilities. A trimester academic year, with 2 entering class sections per year, will go into effect with completion of the new Basic Sciences Building. The wide selection of course and career electives and a scholarly concept of the personal nature of medical care form this school's design for medical education.

John Parks, M.D.
Dean, George Washington University Medical Center

How to Read a Speech*

Under most circumstances the typical audience situation is far from ideal. And particularly in this frenzied 20th century living it is difficult to cut through audience lethargy and distractions to make listeners hear, understand and respond. To be successful, the speaker must attain impressiveness.

The speaker will be impressive in delivery when he has:

1. Authority
2. Involvement
3. Communicativeness
4. Vocal expressiveness
5. Physical expressiveness

The speaker will attain authority, communicativeness and involvement when he is confident, direct, enthusiastic, friendly and genuine.

He should wring the most out of his vocal pitch, rate and loudness by achieving variety, avoiding extremes, and causing the variety to be generated by context. Improvement of vocal quality or timbre so that it is resonant and mellifluous may require clinical attention, especially if the speaker has quality that is breathy, excessively nasal or denasal, husky, harsh, strident or metallic. But, happily, if the speaker effectively modulates his pitch, rate and loudness, this not only will compensate for mediocre or inferior timbre, but in a great many cases will improve it.

The speaker will remind himself that perhaps the greatest single danger with regard to voice is speaking too quietly. Other vocal habits may be annoying, but the inability to be heard is downright insulting. The speaker will experiment with the public address system in advance to be sure that he is using it properly. If this is not possible he will watch carefully his predecessors at the microphone. Most important, if there is any doubt he will simply ask the members of the audience outright: "Can you hear me?" "Am I too loud?" "Is this about right?"

The speaker will achieve physical expressiveness by employing movement about the platform; posture that is erect and comfortable; gesture of hands, head, torso; animated and friendly facial expression; and eye contact that approaches as nearly as possible 100 per cent of the time and contact with all members of the audience. This bodily action will be plentiful, meaningful and spontaneous.

The speaker will remind himself that physical

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* Editor's note: This is the summary, reprinted by permission, from a booklet by the same title, prepared by Robert Haakenson, Ph.D., of the Department of Public Relations, Smith Kline & French Laboratories, Philadelphia. The Executive Office of the Medical Society has on hand a number of copies of this booklet, delightfully illustrated, available upon written or telephone request (223-2230).

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expressiveness is valuable for the audience because it attracts attention and holds interest and is very helpful in transmitting meaning. He will also remind himself that physical expressiveness is perhaps even more valuable for him, the speaker, because it serves as a springboard to project him into a spirit of total communicativeness.

Three principles for effectiveness in practice and drill work are:

1. Complete familiarization with speech and speaking circumstances;
2. Overdoing vocal and physical expressiveness in rehearsal; and
3. Carrying over rehearsal techniques into everyday speech.

Specific techniques for readying a given speech for delivery include the succession of steps that lead progressively from simple silent reading to presenting it in circumstances that simulate as exactly as possible the actual speaking occasion, including an "audience" and critics.

Preparation for delivering a speech by reading can be as enjoyable as it is rewarding if the speaker will allow himself time, set a reasonable goal (not too modest), follow the steps outlined above systematically and thoroughly, and resolve that he is going to make his best possible effort to evoke from the audience the response he wants.

Proposal for a Pilot Survey on Leukemia

The medical profession is fortunate to have the moral and financial support of many highly intelligent and strongly motivated laymen. This situation is especially true of the Greater Washington Chapter of the Leukemia Society of America, Inc. The lay members and the medical members of the local and the national medical advisory boards have approved the conducting of a survey of cases of leukemia and related diseases in this area.

The objective is to accumulate information on the items included in the survey questionnaire, which is reproduced on the accompanying pages. These items are self-explanatory.

It is known that over 400 new cases of leukemia are diagnosed in this area each year, yet the information available about these cases is limited. The information will be kept CONFIDENTIAL. Each case will be assigned a number and, where available, the Social Security number will also be recorded.

The information will assist the Leukemia Society with its Patient Aid Program and will assist the physicians by providing a feed-back on the scope and management of leukemia in this area.

The survey will be successful only if 100 per cent of the leukemia cases are counted. All costs of the questionnaire form and mailing will be borne by the Washington Chapter of the Leukemia Society of America, Inc., 1801 Eye Street, N.W., Washington, D. C. 20006 (telephone: 628-0795). The questionnaire will be obtainable from this office as well as from hospital record rooms and from medical societies of the area. The completed forms should be placed in the postage-paid envelope provided, which is pre-addressed to the Washington Medical Center, Inc., Data Processing Center, 915 19th Street, N.W., Washington, D. C. 20006. This Center has been designated the Leukemia Accumulation Information Retrieval Center (LAIR). The questionnaire will also be obtainable from the Center.

George P. Blundell, M.D.
Chairman, Medical Advisory Board,
The Greater Washington Chapter of
The Leukemia Society of America, Inc.

Woman's Auxiliary Officers for 1968–69 (page 624)
Physicians are urged to take this issue home to their wives.

LEUKEMIA SOCIETY OF AMERICA INC.
ACCUMULATION INFORMATION RETRIEVAL CENTER

WASHINGTON MEDICAL CENTER, INC.
DATA PROCESSING CENTER
915 19th STREET, N.W.
WASHINGTON, D. C. 20006

The Greater Washington Chapter of the Leukemia Society of America Inc., is conducting a survey of leukemia cases in the Greater Washington area. It is authorized by the National Office and supervised by the Medical Advisory Board whose members include:

George P. Blundell, M.D., Chairman
William G. Battaile, M.D.
C. Barrie Cook, M.D.
William D. Dolan, M.D.
Oscar B. Hunter, Jr., M.D.
Sanford L. Leikin, M.D.
William L. Marsh, M.D.
Kenneth L. McCoy, M.D.
Richard E. Palmer, M.D.
Charles E. Rath, Jr., M.D.
William C. Swatek, M.D.
C. Gordon Zubrod, M.D.

The Leukemia Society Accumulation Information Retrieval Center (LAIR) is designed to aid the local chapter in the effective programming of its activities. They include support to the efforts of the medical profession for research and diagnosis, as well as assistance to the patient.

The Center is undertaking a one-year pilot study. The cooperation of all the physicians of the Greater Washington area is requested for the completion of this form on all new cases of diseases of the reticulo – endothelial system. 100% recording of cases is necessary for the success of this program.

These forms are available from The Washington Chapter, Leukemia Society of America, Inc., 1801 Eye Street, N.W., Washington, D. C. 20006, telephone 628-0795, as well as hospital record rooms and area medical societies. Pre-paid postage – self addressed envelopes accompany each form.

CONFIDENTIAL

This information is kept **CONFIDENTIAL**

PATIENT ID
NUMBER:
ASSIGNED BY LEUKEMIA SOCIETY

Contributing Physician: last name first initial	Phone

Physician Address:
street city state zip

Patient Present Address: street city state	Phone zip
Place of Employment: street city state	Phone zip

Patient Previous Address (within last 4 years):
street city state zip

Hospital Address:
street city state zip

Date of Birth: year month day	Place of Birth: city state	Sex: 1. Male 2. Female	Race: 1. Caucasian 2. Afro American 3. Other

Occupational Status:
1. Active 2. Semi-retired 3. Retired 4. Housewife 5. Schoolchild 6. Deceased

Marital Status: 1. Married 2. Single	Occupation:

Income Level When Illness Was Discovered:
1. $0 – $4,000 2. $4,000 – $6,000 3. $6,000 – $8,000 4. $8,000 – $10,000 5. Over $10,000

Social Security Number:

Date of Diagnosis	Date of Onset (Est.)	No. of Times Hosp.	Total Mos. Hospitalized

If Deceased, Was Necropsy Performed?
1. Yes 2. No
If Yes, Where?

This information is kept **CONFIDENTIAL**

Please circle appropriate letter(s) or number(s)

THE PATIENT HAS (HAD):

- a. Acute myelogenous leukemia
- b. Chronic myelogenous leukemia
- c. Acute lymphatic leukemia
- d. Chronic lymphatic leukemia
- e. Monocytic leukemia, Naegeli type
- f. Monocytic leukemia, Schilling type (histocytic)
- g. Agnogenic myeloid metaplasia
- h. Mast cell leukemia
- j. Plasma cell leukemia
- k. Multiple myeloma
- l. Hodgkins Disease, Paragranuloma
- m. Hodgkins Disease, Granuloma
- n. Hodgkins Disease, Sarcoma
- p. Lymphosarcoma
- q. Reticulum cell sarcoma
- r. Erythroleukemias (Di Guglielmo's syndrome)

TECHNIC OF DIAGNOSIS:

1. Number of bone marrow exams
2. Lymph node biopsy
3. Skin biopsy
4. Other

THERAPY USED:

a. Drugs:
  1. Leukeran
  2. Myleran
  3. ThioTEPA
  4. Mercaptopurine
  5. Methotrexate
  6. Cytoxan
  7. Vincristine
  8. Cortisone
  9. Antibiotics
  10. Other

b. Radiation:
  1. X-ray
  2. Isotopes (P^{32})
  3. Other

c. Blood Transfusions
  1. None
  2. Single
  3. Multiple

d. Platelet Transfusions
  1. None
  2. Single
  3. Multiple

Continued

This information is kept **CONFIDENTIAL**

Please circle appropriate number(s)

- CLINICAL HISTORY OF EXPOSURE TO:

a. Radiation
  1. Single large
  2. Multiple small
  3. Patient's mother during pregnancy
  4. Other:

b. Chemical/Drugs
  1. Benzol
  2. Xylol
  3. Toluol
  4. Arsenicals
  5. Sulfonamides
  6. Hexachlorcyclohexane
  7. Phenylbutazone
  8. Insect repellent
  9. Other

c. Familial History
  1. Mother
  2. Father
  3. Brother
  4. Sister
  5. Grandparent
  6. Other

d. Family Pets
  1. Dog
  2. Cat
  3. Bird
  4. Other

- DURATION OF SYMPTOM PRIOR TO DIAGNOSIS: ______________
  Year Month Day

- CLINICAL COURSE:
  Number of relapses ______________ Number of remissions ______________

- COMPLICATIONS:
  1. Thrombocytopenia
  2. Bacterial infection
  3. Fungal infection
  4. Viral infection
  5. Other infection
  6. Splenomegaly
  7. Hepatomegaly
  8. Lymph nodes
  9. Anemia

Remarks:

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Special Program on Drugs and Drug Abuse

during the Medical Society's Annual Scientific Assembly at the Hotel Statler-Hilton

Friday, November 22, 9:00 a.m.

Comparative Performance of Equivalent Drugs

Moderator: **C. Willard Camalier, Jr., M.D.**

Panelists:

Gerhardt Levy, Pharm.D., Chairman, Department of Pharmaceutics, State University of New York at Buffalo
William Murray, M.D., Bureau of Medicine, Food and Drug Administration
C. Joseph Stetler, President, Pharmaceutical Manufacturers Association

Drug Abuse: Problems as Viewed by the Law Enforcement Officer, Judiciary, Investigator, Community Health Officer, and Medical Clinician

Moderator: **Seymour Alpert, M.D.**

Panelists:

Mr. Joseph Arpaio, Agent in Charge, Federal Bureau of Narcotics and Dangerous Drugs, Washington Field Office
The Honorable William B. Bryant, U. S. District Judge for the District of Columbia
Nathan Eddy, M.D., Consultant on Narcotics and Drug Abuse, NIH
Murray Grant, M.D., Director of Public Health, District of Columbia
Sherman Kieffer, M.D., Associate Director for Patient Care, NIMH

The Royal Society of Medicine, London*

The Royal Society of Medicine, under the name of the Medical and Chirurgical Society of London, was founded on 22 May 1805 at a meeting in the Freemasons' Tavern of the leading London physicians and surgeons of that time. They agreed "that a Society comprehending the several branches of the medical profession be established in London for the purpose of conversation on professional subjects, for the reception of communications, and for the formation of a library."

The Society's first home was at No. 2 Verulam Buildings, Gray's Inn. By 1810 larger premises were needed, and growth continued during the next 24 years when the Society moved 3 times, on each occasion to larger premises within Lincoln's Inn Fields.

By 1834 the development of the library and the increase of the Society's influence on science and medical practice had become of such importance that it received from King William IV a Charter of Incorporation as The Royal Medical and Chirurgical Society of London.

This was a significant period not only in the Society's history but also in the history of medicine and medical societies generally for, during the succeeding years, many medical societies were formed for the advancement of their own specialty. This gradual dissemination of specialist knowledge continued throughout the latter half of the 19th century in spite of many attempts to unite the various societies in one body representing every department of medicine and surgery. The Royal Medical Chirurgical Society campaigned actively to bring this about. By a Supplemental Charter granted by King Edward VII in 1907, the original charter was enlarged, the Society's name was changed to The Royal Society of Medicine, and it was empowered to form Sections and to admit the members of other societies which agreed to dissolve and to amalgamate with it. Seventeen societies dissolved at that time and were reformed as Sections representing their own specialties.

At this time the Society's house was at 20 Hanover Square, and it was obvious, soon after this amalgamation, that new and much larger premises ought to be acquired. Consequently, in 1912 the Society moved to a completely new building, designed for its purposes and "of a character consistent with the importance of its objects and with the greatness of the metropolis of the Empire." It is this building, at 1 Wimpole Street, which is the Society's present headquarters, though it was enlarged in 1952 and again in 1964. In 1912 there were just over 3,000 members served by a staff of 12; today, there are 13,000 members requiring a staff of 130. Then, there were 80,000 bound volumes; now there are 500,000 volumes. From 17 Sections, holding 140 meetings, there are 32 Sections holding approximately 250 meetings; in addition, 250 meetings of other societies are held on the premises each year.

Chandos House was acquired in 1964, and its Mews was converted into a Domus Medica, providing overnight accommodation for provincial and overseas members and for visiting speakers from all parts of the world. A large bookstore was built in the basement of Chandos House, and some of the lesser used books and journals are now stored there. Other parts of Chandos House are in use as offices, while the great drawing rooms are used for additional meetings, receptions, and ceremonial occasions, and for a regular buffet luncheon service.

This expansion has been dictated by the obligation which the Society has under its charters to provide academic services for "the cultivation and promotion of physic and surgery and of the branches of science connected with them." Its activities range over the whole of the clinical and preclinical field and, in addition to its Sections of Medicine, Surgery, Obstetrics and Gynecology, and General Practice, it has 28 other Sections, including those of Measurement in Medicine, Occupational Medicine, Odontology, Comparative Medicine, Medical Education and, most recently formed, Plastic Surgery.

The meetings of the Sections provide a forum for the presentation of papers and for discussion, perhaps unequalled anywhere in medical circles. They are reported in the monthly publication, the *Proceedings of the Royal Society of Medicine*, which is distributed to every member of the Society. This journal also carries reports on the national and international symposia which the Society and the Sections organize each year. The Society's library is one of the largest and most comprehensive medical libraries in the world, and certainly the most comprehensive outside the United States of America, receiving over 2,300 current periodicals each year. Another service provided by the Society is the maintenance of a Photographic and Film Unit which undertakes most kinds of medical documentation, photography and cinematography for doctors, institutions and universities throughout the world. The Society also provides meeting rooms for homeless medical and scientific societies, club and restaurant services for members using the Society's House, and overnight accommodation for distant members.

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* Prepared for the Medical Annals by Mr. R. N. Thompson, Executive Assistant, and submitted by Mr. R. T. Hewitt, Secretary, The Royal Society of Medicine, 1 Wimpole Street, London, W. 1.

Because of the comprehensive nature of the Society, it is well suited to make a contribution, not only in an interdisciplinary capacity in this country, but in the international field. Every encouragement is given to promoting formal and informal exchanges between the Society and visiting specialists and specialist societies abroad. This is done through a growing overseas membership, through the Society's Sections, and through the establishment of reciprocity with similar learned societies overseas. Societies in this category are the Academy of Medical Sciences, Andhra Pradesh, Hyderabad; The Royal Australasian College of Surgeons, Melbourne; the Dansk Medicinsk Selskab, Copenhagen; The New York Academy of Medicine; the Norwegian Medical Society, Oslo; the Collège de Médecine des Hôpitaux de Paris; the Swedish Medical Society, Stockholm; the Academy of Medicine, Toronto, and the Union of Scientific Medical Societies in the Socialist Republic of Romania, Bucarest. It is particularly gratifying to be able to record that a similar arrangement has recently been entered into with the Medical Society of the District of Columbia.

It is always the Society's wish that such arrangements should extend beyond a formal agreement to a practical and lively exchange of courtesies. Because of this it is agreed that members of societies with which reciprocity has been established should have the free use of this Society's House when they are in London, with admission to any meetings being held here, access to the library for reading, and the use of the club rooms and restaurant. The staff of the Society also gladly undertake to give these visiting members any possible help in the way of introductions to colleagues in this country.

These activities were given fresh impetus when, as a result of recommendations made by the medical committee formed in the United Kingdom during International Coöperation Year, 1965, the Society established an International Office devoted to promoting and fostering relations between members of the medical profession in this country and overseas. This office has the task of collecting information from all parts of the world which is likely to be of interest and help to medical men travelling abroad, and it is readily available to enquirers.

Another great advance in the international field has been the recent establishment, in New York, of the Royal Society of Medicine Foundation, Inc., with the purpose of strengthening coöperation and understanding between members of the medical profession in the U.S.A. and in the United Kingdom. This the Foundation will do either by contributing toward the costs of the Society's current activities in this field or by itself promoting activity of this kind in collaboration with the Royal Society of Medicine.

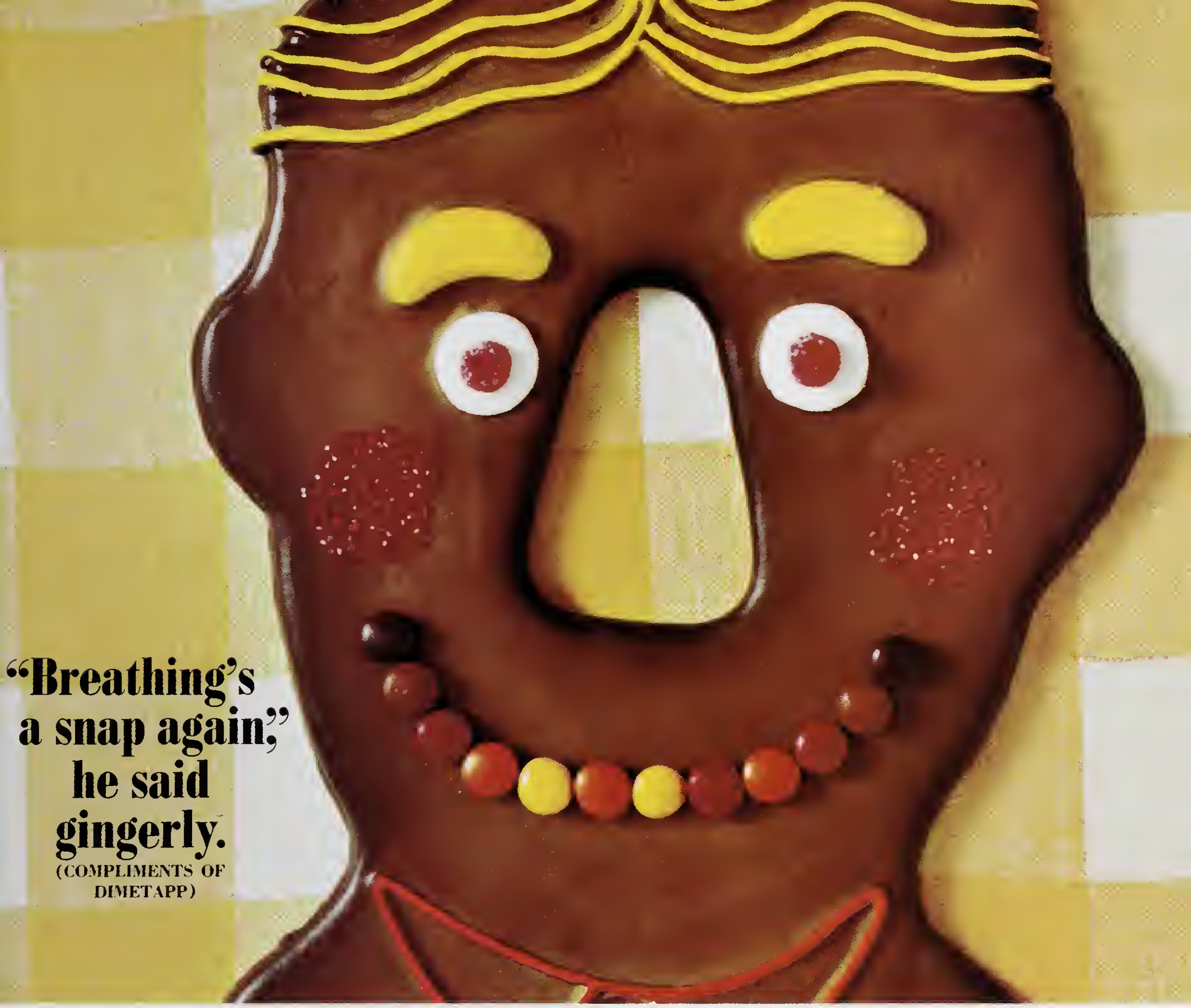
The above activities, and others which are under consideration by the Society, provide opportunities for the Society's International Relations Office and for the Foundation in New York to play a full part in a program which has widened immeasurably since those early days in 1805, probably to an extent far beyond the imagination of the Society's founders. It is unlikely that they foresaw a Society with links beyond the seas, which could contribute to and benefit from medical science throughout the world. Yet the Society's present activities derive from the original intentions of the founders who established a Society dedicated to the "cultivation and promotion of physic and surgery."

Invitation to History-minded Physicians

The Washington Society for the History of Medicine was formed in February, 1961 by a group of individuals in the Washington Metropolitan Area who shared an interest in the history of medicine and allied sciences. The Society, an affiliate of the American Association for the History of Medicine, meets bimonthly, except during the months of June, July and August, and attempts to present an interesting and varied program of historical papers. The speakers include members of the local society as well as outstanding historians who are invited as guest speakers at least twice a year. Previous guest speakers have included Owsei Temkin, Rene Dubos, Lloyd Stevenson, and Lester King.

The bimonthly meetings, which begin in September, are held in the Billings Auditorium of the National Library of Medicine. Interested persons are requested to contact the Secretary-Treasurer, Dr. Peter D. Olch, whose address is the History of Medicine Division, National Library of Medicine, 8600 Rockville Pike, Bethesda, Maryland 20014 (telephone: 496-5961). Annual dues for the Society are $3.00.

William C. Roberts, M.D.
President, Washington Society
for the History of Medicine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LOW-BACK PAIN

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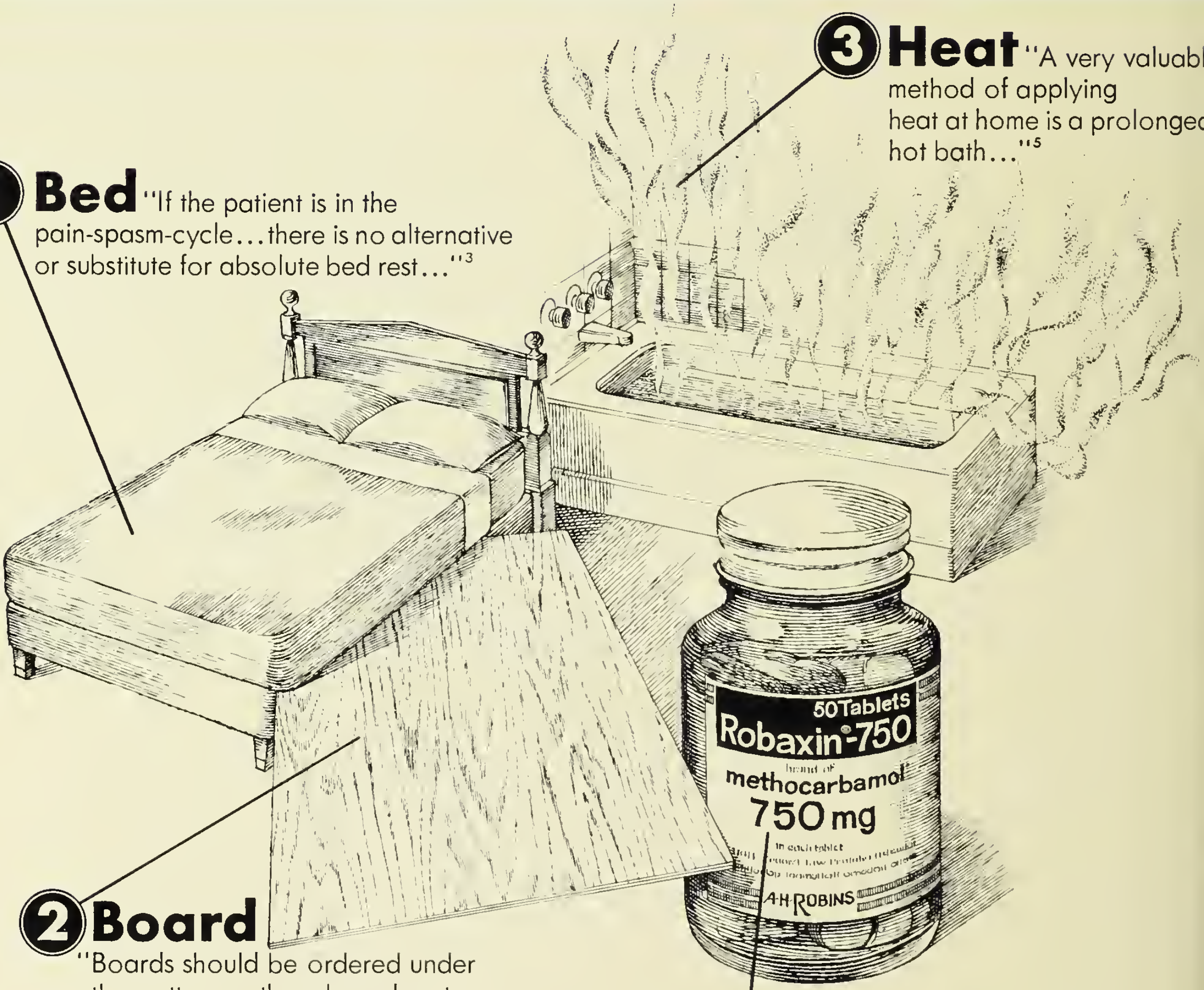

③ **Heat** "A very valuabl method of applying heat at home is a prolongec hot bath..."[5]

① **Bed** "If the patient is in the pain-spasm-cycle...there is no alternative or substitute for absolute bed rest..."[3]

② **Board** "Boards should be ordered under the mattress...these boards act by immobilizing the spine..."[4]

④ **Robaxin®-750** (methocarbamol, 750 mg. capsule-shaped tablets) A well-tolerated[6] skeletal muscle relaxant, methocarbamol helps relieve spasm "...without interfering with normal tone and movement."[7] And there is little likelihood of sedation.[6]

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► HEALTH DEPARTMENT NOTES

Tri-level Health Plan for D.C.

A NEW TRI-LEVEL HEALTH PLAN for the District of Columbia consists of neighborhood health centers, an area health center, and D. C. General Hospital.

Six Neighborhood Health Centers which provide 1-stop family health care to holders of D. C. Medicaid or to other District residents eligible for services under Health Department programs have opened, 5 of them in existing Health Department facilities.

Through a new program health services are provided at 3 levels—in neighborhood health centers, area health centers, and hospitals. Planning and implementation of the tri-level health plan and staffing and equipping the health centers have been accomplished without any additional personnel or funds.

Neighborhood Health Centers are located in the Parkside Health Clinic, 701 Kenilworth Terrace, N.E.; Health Clinic, 635 H Street, N.E.; Potomac Gardens, 13th and G Streets, S.E.; Southwest Health Center, Delaware Avenue and I Streets, S.W.; Arthur Capper Clinic, 1011 7th Street, S.E.; and 941 North Capitol Street. Each Center will serve a population of about 25,000.

The Area Health Center is located at the present Area C Community Health Center on the grounds of D. C. General Hospital. Two new Health Centers for Mothers and Children at 702 15th Street, N.E., and 4130 Hunt Place, N.E., supplement the Area Health Center in offering care to mothers and children.

D. C. General Hospital is used for the hospital level of care and treatment.

Services provided by the Neighborhood Health Centers include health tests and medical evaluations, immunizations, and health care for sick children and adults. A basic medical history is obtained for each patient, and most medical care offered is of a general nature, including uncomplicated pediatrics, prenatal and postnatal care, family planning guidance, and general treatment of minor illnesses. When additional diagnosis or treatment is needed the patient is referred to the Area Health Center. Transportation is furnished as much as is possible.

With a staff of about 20, the Neighborhood Center will serve approximately 25,000 neighborhood residents. Heading the staff are physicians, one of whom is an internist or a general practitioner, one a pediatrician, and another an obstetrician-gynecologist. Other staff members are nurses, health aides, nutritionists, medical social workers, and other health specialists.

A nurse-family relationship has been established at each Neighborhood Center. Members of each family see the same registered nurse at each visit, and after consultation the nurse determines which physician the patient should see.

The Area Health Center provides dental screening and treatment service and a full range of inpatient, outpatient, and day-care services in mental health and mental retardation, including some services for alcoholics and drug addicts.

The Area Center contains a laboratory and is the center for health activities covering a section of the city. The Area Center also has general pediatrics, medical (including tuberculosis and venereal diseases), general surgical, obstetrics and gynecology, and birth control clinics.

In addition to being a district office for public health nurses the Area Center serves as a headquarters for other health personnel working in that part of the city. It has supporting services in nutrition, speech and hearing, physical and occupational therapy, and medical social service, as well as coördinators for health activities such as mental health, laboratory services, and health education.

D. C. General Hospital continues to offer specialized inpatient and outpatient services and emergency treatment to patients referred there by either the Neighborhood or Area Health Center physician.

Citizens' advisory committees composed of 25 persons each work closely with the Neighborhood Health Centers. They make known to the Center Health Directors the health needs within the neighborhood, advise on the hours and days of operation of the Center, and encourage people in the neighborhood to visit the Center and utilize the health services it provides.

The Tri-level Health Program is a pilot project. It is ultimately planned to have from 6 to 10 Neighborhood Health Centers in each of the 4 health areas of the city. These in turn will be served by an Area Health Center and have access to a base hospital.

MURRAY GRANT, M.D., D.P.H.
D. C. Director of Public Health

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NEW MEMBERS

Active

Hubert Joseph Alpert, M.D. Office: 1234-19th Street, N.W. Born in Chicago, Ill. Medical degree: University of Illinois, 1962. Specialty: Internal Medicine.

Myles Howard Clark, M.D. Office: 806 Alabama Avenue, S.E. Born in Jackson, Miss. Medical degree: Howard University, 1965. Specialty: Pediatrics.

Michael Romollo Cuadra, M.D. Office: D. C. General Hospital. Born in the Philippines. Medical degree: University of Santo Tomas, The Philippines, 1958. Specialty: Pathology.

Stuart Henry Danovitch, M.D. Office: 110 Irving Street, N.W. Born in Cambridge, Mass. Medical degree: Northwestern University, 1960. Specialty: Internal Medicine.

Eduardo Reyes Ilano, M.D. Office: Sibley Memorial Hospital. Born in Manila, The Philippines. Medical degree: University of the Philippines, The Philippines, 1961. Specialty: Internal Medicine.

Richard Joseph Kowalski, M.D. Office: 5301 Macomb Street, N.W. Born in Ithaca, N. Y. Medical degree: Georgetown University, 1964. Specialty: Psychiatry.

Hunter Ellsworth Malloy, M.D. Office: 19th and E Streets, S.E. Born in Portsmouth, Va. Medical degree: Meharry Medical School, 1963. Specialty: Otolaryngology.

John Frederick Mermel, M.D. Office: 2141 K Street, N.W. Born in Denver, Colo. Medical degree: George Washington University, 1960. Specialty: Internal Medicine.

Allen M. Mondzac, M.D. Office: 2300 K Street, N.W. Born in Washington, D. C. Medical degree: George Washington University, 1961. Specialty: Internal Medicine.

Robert Michael Nedzbala, M.D. Office: 2201 C Street, N.W. Born in Jersey City, N. J. Medical degree: Georgetown University, 1962. Specialty: Internal Medicine.

Ramasamynatesan, M.D. Office: 922 East Capitol Street. Born in Kodumudi, India. Medical degree: Madras Medical College, India, 1960. Specialty: Psychiatry.

Massimo Alberto Righini, M.D. Office: 2010 R Street, N.W. Born in Florence, Italy. Medical degree: University of Bologna, Italy, 1958. Specialty: General Surgery.

Richard Alan Schwartz, M.D. Office: 1100 Sixth Street, S.W. Born in Saint Clair, Mich. Medical degree: Cornell University, 1965. Specialty: General Practice.

Naren N. Shah, M.D. Office: 2121 Pennsylvania Avenue, N.W. Born in Ahmedabad, India. Medical degree: B. J. Medical College, India, 1957. Specialty: Internal Medicine.

Michael Mendel Shefferman, M.D. Office: 2141 K Street, N.W. Born in Baltimore, Md. Medical degree: University of Maryland, 1962. Specialty: Internal Medicine.

John Chandler Smith, M.D. Office: 1335 H Street, N.W. Born in Denver, Colo. Medical degree: University of Oregon, 1944. Specialty: Pathology.

Mark On-Man Tso, M.D. Office: Armed Forces Institute of Pathology. Born in Hong Kong. Medical degree: University of Hong Kong, Hong Kong, 1961. Specialty: Ophthalmology.

Frances L. Cress Welsing, M.D. Office: 1846 Park Place, N.W. Born in Chicago, Ill. Medical degree: Howard University, 1962. Specialty: Psychiatry.

Lennox Samuel Westney, M.D. Office: 65 Massachusetts Avenue, N.W. Born in Kingston, Jamaica, W. I. Medical degree: Howard University, 1961. Specialty: Obstetrics and Gynecology.

Associate

Raymond L. Font, M.D. Office: 700 Hermleigh Road, Silver Spring, Md. Born in Matanzas, Cuba. Medical degree: University of Havana, Cuba. Specialty: Pathology.

Roberta Ann Irish, M.D. Office: Sibley Memorial Hospital. Born in Lorain, Ohio. Medical degree: Western Reserve University, 1957. Specialty: Ophthalmology.

Arthur V. Mitchell, M.D. Office: 4682 South 34th Street, Arlington, Va. Born in Methuen, Mass. Medical degree: Georgetown University, 1936. Specialty: General Practice.

Baboo Jayantilal Rajvaidya, M.D. Office: Rockland State Hospital, Orangeburg, N. Y. Born in Ahmedabad, India. Medical degree: B. J. Medical College, India, 1961. Specialty: Psychiatry.

Resident-Intern

George Z. Weissfisch, M.D. Office: 110 Irving Street, N.W. Born in Bratislava, Czechoslovakia. Medical degree: University of Guayaquil, Ecuador, 1964. Specialty: Surgery.

Patients Needed at Clinical Center

The coöperation of physicians is requested in the referral of (1) patients for a continuing study of hormone-producing tumors of the testes and (2) patients with metastatic carcinoma of the breast, both studies being conducted by the Endocrinology Branch of the National Cancer Institute at the Clinical Center, National Institutes of Health, in Bethesda, Md.

For the first study patients with choriocarcinoma of the testes are needed, particularly those having high titres of gonadotrophins or positive pregnancy tests. Patients of all ages will be considered for endocrine studies and chemotherapy.

Particularly needed for the second study are patients with metastatic carcinoma of the breast who have had no previous endocrine therapy. Patients who are premenopausal, and candidates for surgical oöphorectomy, or postmenopausal, and candidates for hormonal treatment, will undergo endocrine studies prior to and after endocrine therapy.

Physicians who wish to have their patients considered for these studies may write or telephone Dr. Marvin A. Kirschner, Clinical Center, Room 12N–206, National Institutes of Health, Bethesda, Md. 20014; telephone: (301) 496-3079.

Woman's Auxiliary Notes

The new President of the Woman's Auxiliary to the Medical Society of the District of Columbia, Mrs. Pauline (Polly) Allman Suraci, came to Washington in 1940 from Charlotte, North Carolina, to continue her nurse's training at Sibley Memorial Hospital and Catholic University. At Sibley she met her future husband, Dr. Alfred J. Suraci, who was training in surgery. Their many activities and interests, together with those of their 7 children, make the present home in College Heights Estates, Maryland, a busy place.

Polly is President of the Sibley Hospital Alumni, Vice President of Regina High School Home and School Association, and a member of the Women's Advisory Board to American University. She represented the Woman's Auxiliary to the Medical Society on the planning committee for the Homemakers' Service, which is now financed by the United Givers Fund. She has served as President of the Women's Board of Providence Hospital and of Leonard Hall Naval School Auxiliary.

MRS. ALFRED J. SURACI

President, Woman's Auxiliary

Polly was delighted to learn that the new Secretary of the Medical Society, Mr. Tom Sadler, is a fellow "Tar Heeler" (for the uninformed, a North Carolinian).

Officers who have been elected to serve with Mrs. Suraci are:

President-elect: Mrs. Raymond A. Osbourn
First Vice President: Mrs. Albert E. Marland, Jr.
Second Vice President: Mrs. J. Peter Murphy
Recording Secretary: Mrs. George W. Hyatt
Assistant Recording Secretary: Mrs. George T. Economos
Corresponding Secretary: Mrs. Harold T. Yates
Assistant Corresponding Secretary: Mrs. Thomas M. Foley
Treasurer: Mrs. George H. Crofton
Assistant Treasurer: Mrs. Russell E. Iler

To carry out the motto for this year, "Let's Make Membership More Meaningful," Mrs. Charles V. Pate, Chairman of the Program Committee, has arranged an excellent offering of social, civic, and cultural activities.

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News & Personals

Summary of Executive Board Actions

Proceedings of September 23 Meeting

Dinner for the Board members was served at the Army–Navy Club on Farragut Square. Dr. Edward W. Nicklas, Chairman, presided at the meeting which was attended by Drs. William S. McCune, Frank S. Bacon, Jed W. Pearson, Jr., Gudrun Augustin-Rueckert, Paul R. Wilner, Robert J. Coffey, Henry D. Ecker, John J. Hughes, C. Willard Camalier, Jr., and Mr. Thomas Sadler, Secretary. Present by invitation was Dr. Murray Grant, D. C. Director of Public Health. The meeting was called to order by Dr. Nicklas at 7:55 p.m.

The minutes of the August 26, 1968 Board meeting were approved as submitted.

Committee Meetings: The Secretary reported the following committees had met since the last meeting of the Board:

August	29	Traffic Committee
September	4	Fee Review Committee
	5	Insurance Committee
	5	Committee on Communicable Diseases
	10	Board of Credentials
	10	Committee on Public Information and Education
	11	Membership Committee
	17	Committee on Liaison with the Clergy
	17	Grievance Committee
	18	House and Building Committee
	19	Traffic Committee
	20	Committee on Constitution and By-laws

Board of Credentials: On recommendation of the Board of Credentials, 19 applicants were elected to Active membership, 4 to Associate membership, and 1 to Resident-Intern membership. Their names appear on page 623. Approval was given to the following changes in membership status:

Transfers

Active to Special: Joel N. Novick and Daniel B. Washington

Active to Associate: Margaret E. Callan, Liselotte Hochholzer, Laurence E. Rubin, Paul D. Sullivan, Jr., and David Arthur Zohn

Active to Resident-Intern: Lee W. Davis

Resident-Intern to Active: John J. Niles, Jr.

Reinstatement

Active: Dimitr Christov

Resignations

Active: Walter Highman Fanburg

Associate: Donald Pettus Conwell and John H. Edgcomb

Establishment of an Awards Committee: Upon the recommendation of the Membership Committee that an Awards Committee be established on a permanent basis, the Board authorized the President to appoint such a committee.

Plaque for Recognizing Assistance in Acquiring and Building New Headquarters Building: There was much discussion of the House and Building Committee's proposal for a plaque to honor all members who were instrumental in acquiring the land as well as those who helped to guide the construction of the building, provided its furnishings, etc.

The Board agreed with the recommendation and voted that the plaque be prominently displayed in the Society's headquarters, that no specific individuals be named but that there be a broad encompassing statement covering all those who participated.

Neighborhood Health Centers: At the request of the Executive Board, Dr. Murray Grant was invited to review again the Neighborhood Health Centers. The information he presented was essentially the same that he gave at the March 25, 1968 meeting of the Board. Dr. Grant stated that the program is just getting under way and he hopes to review its progress with the Board so that recommended changes can be effected.

Luncheon Meeting with Dr. Grant: Dr. McCune summarized his September 23 luncheon meeting with Dr. Grant. The latter reported that 225 D. C. physicians had signed up for Medicaid and that he was optimistic about more names

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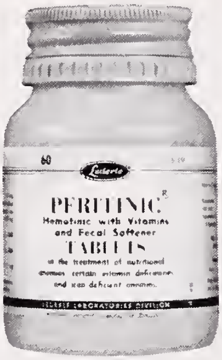

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being added to the roster. As to the licensure law, he submitted to Dr. McCune a copy of suggested amendments to the Healing Arts Practice Act which had been prepared in his office. (It was agreed that copies of these proposed amendments be circulated to the Executive Board for discussion at the next meeting and that copies also be sent to members of the Ad Hoc Committee on Licensure.)

Dr. Grant stated that optometrists were expressing a desire to practice in hospitals. Dr. McCune suggested to him that he query the local hospitals by letter as to whether their staff ophthalmologists are able to meet their needs in the eye care area before proceeding further.

Community Health Week: Mr. Sadler reported that Community Health Week will be observed at Society headquarters, October 20–23. During that time free tests (blood, diabetes, ECG, chest X-ray, etc.) will be made available to the public. He reported also that a Kickoff Jog was being planned for Sunday, October 20, with 25 or more physicians, led by Senator William Proxmire, jogging from the Washington Monument to the Society headquarters.

Cocktail Party for Press, TV and Radio: Dr. McCune announced that a cocktail party would be held for press, radio, and TV personnel and officials on November 7 at the Cosmos Club. The Board agreed that this was an excellent idea but suggested that the Society's representatives at the party be limited to those concerned with the Assembly.

Other Business

1. The Board approved Mrs. Philip Steptoe's request for a party in the Exhibit Hall on a Saturday night in December for the Woman's Auxiliary to the Student American Medical Association of Howard University.

2. A recommendation of the Committee on Communicable Diseases regarding booster vaccinations for children prior to registering for their first school year was approved.

3. The following recommendations of the Committee on Constitution and By-laws were approved:

(a) That Chapter IX—Councils and Committees of the Constitution and By-laws be repealed.

(b) That the 4 Standing Committees remain, namely, Constitution and By-laws, Classification, Grievance, and Medical Ethics and Professional Conduct.

(c) That other committees may be created by the Executive Board to carry out the purposes and business of the Society.

4. A motion was made and carried that the Newsletter be made available for listing changes in the practice status of member physicians.

5. The 3 nominees submitted by Dr. McCune for appointment to the Board of Trustees of Medical Service were approved. The nominees are Drs. Charles E. Bickham, Edward W. Nicklas, and John A. Kenney, Jr.

There being no further business, the meeting was adjourned at 10:30 p.m.

MEETINGS

The annual **John Fitzgerald Kennedy Memorial Lecture,** sponsored by the Department of Pediatrics of Georgetown University, will be given at 10:30 a.m., November 22, in the Gorman Auditorium at the Georgetown Medical Center. The lecturer this year is Dr. Irving Schulman, Professor and Chairman of the Department of Pediatrics of the College of Medicine at the University of Illinois. His subject will be "The Physician—The Reluctant Warrior." Grand rounds concerned with hematological problems, Dr. Schulman's special interest, will be conducted at 8:30 a.m. preceding the lecture. All interested physicians are cordially invited to attend the grand rounds and the lecture.

Physicians are reminded again that the **American Medical Association** is holding its **22nd Clinical Convention** in Miami Beach, Fla., December 1-4, at the Miami Beach Convention Hall. There will be 125 exhibits reflecting the latest developments in pharmaceuticals, medical equipment, and scientific endeavors. Special postgraduate courses will be offered in diabetes, fluid and electrolyte balance, and thyroid disease. In addition, there will be about 30 medical motion pictures and scientific television programs, live and in color.

The AMA Clinical Convention is designed primarily for the man in practice. Every physician is urged to take advantage of this educational opportunity.

GENERAL NEWS

The services of **Dr. F. J. L. Blasingame, Executive Vice President of the American Medical Association,** have been terminated, according to an announcement, September 9, made jointly by Dr. Blasingame and the AMA Board of Trustees following a meeting of the Board in Chicago.

Dr. E. B. Howard, who has been Assistant Executive Vice President since 1948, was named **Acting Executive Vice President.**

Dr. Blasingame left private practice in Texas to assume his AMA position in 1958. Previously he had been on its Board of Trustees. He is Past President of the Texas Medical Association.

At the same meeting the Board of Trustees authorized retaining a firm of management consultants to conduct an analysis of AMA operations.

On or about July 1, 1969 the **Psychiatric Institute Foundation** will begin a program for retraining currently inactive physicians to help meet community mental health needs. The program has the endorsement and coöperation of the Health Manpower Council of the American Medical Association. Training will be part-time (minimum of 15 hours per week) for 1 year. The curriculum will include basic psychiatry, training in group techniques, and supervision in discrete, subspecialty areas. The latter will include alco-

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holic and addict rehabilitation, child and geriatric psychiatry, and work in community mental health centers. Interested physicians may telephone or write Dr. Jesse Rubin, The Psychiatric Institute Foundation, 2141 K Street, N.W., Washington, D. C. 20037; telephone 223-2700.

Lt. General Kenneth E. Pletcher, USAF, MC, has succeeded Lt. General Richard L. Bohannon, USAF, MC, who retired from active duty last year, as Surgeon General of the U. S. Air Force. Honorary fellowship in the American College of Chest Physicians was conferred upon General Pletcher at the 34th Annual Meeting of the College in San Francisco in June.

The Board of Directors of Doctors Hospital, Inc., has announced the appointment of **General Bohannon** to the position of **Director of Medical Education.**

On August 3, 1968, President Johnson signed into law legislation designating the planned National Center for Biomedical Communications as the **Lister Hill National Center for Biomedical Communications,** honoring the Senator from Alabama. The Center will be a part of the National Library of Medicine, a component of the National Institutes of Health, which Senator Hill and the late President John F. Kennedy helped to create. Preliminary planning calls for a tower-type structure annex to be erected southwest of the Library within the next 4 or 5 years. Senator Hill, an Honorary member of the Medical Society of the District of Columbia, plans to retire at the end of this year.

A Washington Medical-Dental Ski Association is being organized in the Washington Metropolitan Area. Interested physicians and dentists should contact Jules A. Dubit, D.D.S., telephone 864-6535.

OBITUARIES

Oliver Clemence Cox, M.D.

(1887–1968)

Dr. Oliver C. Cox, born in Gilmore County, West Virginia, on November 16, 1887, died, August 2, 1968, in Suburban Hospital after a short illness.

After attending high school in West Virginia, Dr. Cox came to Washington, D. C. in 1907 to begin the study of medicine at the George Washington University. After he received his medical degree in 1911 he served for a year in a rotating internship at St. Elizabeths Hospital. He was licensed to practice in the District in 1911.

During his internship Dr. Cox already had shown a primary interest in surgery. Accordingly, along with his early practice of medicine in the District of Columbia he also trained in surgery under the preceptorship of Dr. Charles Stanley White, from 1913 to 1917. His practice was interrupted in 1917 by a period of service in the United States Army, from September 1917 to December 1918. It is an interesting side note that he was one of the medical officers assigned to the American Task Force to go to Siberia to join an European unit to resist the Communists' takeover in that area. However, when it was decided not to send this military task force Dr. Cox was demobilized from the Army in December 1918. He returned to civilian life, taking postgraduate surgical training at both Northwestern and Tulane Universities in 1918.

In Washington he built a large practice in general surgery, with operating privileges in all the private hospitals. Because of his reputation for removing the vermiform appendix through a small incision, he became known to his patients and colleagues as "Buttonhole Cox." With his general advocacy of small incisions, he influenced the practice of surgery in the District of Columbia.

Dr. Cox was many years ahead of the main stream of surgery in early ambulation, which he advocated in the late twenties. He was the author of a book on surgical techniques, entitled "Atlas of Practical Incisions." He independently discovered the procedure of hysterocolpectomy, but subsequently, on searching the literature to publish a paper, he found that it had already been described. In his own contribution, which was published in the *Annals of the Sibley Medical Association* in May 1958, he included the history of the procedure. He subsequently made a movie of this operation which has been viewed widely, both at local and national meetings. He contributed many articles for local publication, and in 1960 he received the Sibley Medical Association Award for years of meritorious service to and for Sibley Memorial Hospital. He was the first surgeon so honored.

He was elected to Active membership in the Medical Society of the District of Columbia on March 6, 1912, and became a Life member in 1953. He was a member of the American Medical Association.

Dr. Cox neither smoked nor drank and in later years berated his patients for these vices. He was well liked by patients and referring physicians for his forthrightness and surgical finesse. He was a most charitable individual and helped numerous young surgeons and other physicians get started in their practice. He leaves his wife, Mrs. Grace Cox, of 3115 Nebraska Avenue, N.W., 4 sisters, and 2 nephews who are physicians in the Washington area.

Duane C. Richtmeyer, M.D.

Clapham Price King, M.D.

(1889–1968)

Dr. Clapham P. King, Washington physician for 40 years, died, August 23, 1968. For many years he had been associated in the practice of internal medicine with Dr. William Earl Clark and Dr. Hill Carter in offices at 1835 Eye Street, N.W. He retired from practice in 1961.

Dr. King was born on Weston Farm in Harwood, Maryland, July 12, 1889. He attended St. John's College in Annapolis and graduated from the University of Pennsylvania School of Medicine in 1912. He interned at Garfield Memorial Hospital for a year and spent a year as resident physician at Casualty Hospital. From 1915 to 1917 he trained in internal medicine at the Mayo Clinic in Rochester and later, from 1919 to 1922, he was on its permanent staff.

During the years of World War I he served with the Serbian Unit of the American Red Cross and overseas with the U. S. Army Medical Corps. He was subsequently decorated by the Serbian Government.

During his long practice he was associate in medicine on the staffs of Doctors and Georgetown Hospitals and the Washington Hospital Center.

Dr. King was elected to Active membership in the D. C. Medical Society in 1923 and became a Life member in 1964. He was a Fellow of the American College of Physicians and a member of the American Medical Association, the Southern Medical Association, the Clinical Club of Washington, and the International Medical Club of Washington. He was a member of St. Albans Church and the University Club.

He leaves a brother, Thomson King, of Baltimore, Md.

William Joseph Graham Davis, M.D.

(1914–1968)

Dr. William J. G. Davis, Washington ophthalmologist, died, August 3, 1968 at Doctors Hospital.

A Washingtonian and the son of an ophthalmologist, Dr. William Thornwall Davis and Mrs. Reneé T. Davis, who survives him, he was born, June 10, 1914. He graduated from the University of North Carolina in 1939 and from the University of Maryland School of Medicine in 1943. He interned at Johns Hopkins Hospital for a year and was a resident physician in ophthalmology at the Cleveland Clinic from 1944 to 1945.

With World War II in progress he enlisted in the Medical Corps of the United States Army and served from 1945 to 1947. He was also medical advisor to the Selective Service Board of Washington, D. C.

Dr. Davis continued his father's practice at 927 Farragut Square, N.W. after he died in 1944, and in 1953 moved to offices at 1632 K Street, N.W. He was Assistant Clinical Professor of Ophthalmology at George Washington University School of Medicine, senior attending ophthalmologist at the Washington Hospital Center, consultant in ophthalmology to Columbia Hospital for Women, and visiting ophthalmologist at Children's, Doctors, and Sibley Hospitals.

Dr. Davis was a Fellow of the American College of Surgeons and a diplomate of the American Board of Ophthalmology. He was elected to Active membership in the Medical Society of the District of Columbia in 1946. He was also a member of the American Medical Association, the Southern Medical Association, the American Academy of Ophthalmology and Otolaryngology, and the Pan American Association of Ophthalmology. He was a Mason and a member of the Army-Navy Club, the American Legion, and the Chevy Chase Club.

In addition to his mother, he is survived by his wife, Mrs. Helen Jennings Davis, of the home, 2805 29th Place, N.W., and 3 brothers, Robert B. and Akin T. Davis, both of Washington, and Sheldon R. Davis, of Lake Worth, Florida.

Robert Massie Page, M.D.

(1895–1968)

Dr. R. Massie Page, who practiced in Washington for nearly 45 years, died, August 27, 1968 at Georgetown University Hospital. He lived at 3721 Massachusetts Avenue, N.W.

A native of Batesville, Va., Dr. Page was born, November 20, 1895. A graduate of the College of William and Mary, he received his medical degree from the University of Virginia in 1921. He interned for a year at the University of Virginia Hospital and was resident surgeon there from 1922 to 1924.

Dr. Page moved to Washington in 1924. He shared offices for many years at both 1150 Connecticut Avenue, N.W., and later at 1835 Eye Street, N.W., with Dr. W. LeRoy Dunn. He was on the staff of the Washington Hospital Center.

He was elected an Active member of the Medical Society of the District of Columbia in 1925 and became a Life member in 1965. He was a member of the American Medical Association and the Osler Society.

Surviving him are his wife, Mrs. Pauline Johnson Page; a son, Robert W. Page, of Cinncinnati, Ohio; and a daughter, Mrs. M. G. Scott, of Annandale, Va.

Carl Goldenberg, M.D.

(1893–1968)

Dr. Carl Goldenberg, a Life member of the Medical Society of the District of Columbia, died, October 1, 1968, at Doctors Hospital. He lived at 4905 Reno Road, N.W.

Dr. Goldenberg was born in Storndorf in Hesse, Germany, February 2, 1893. He was graduated from the University of Frankfurt in 1920 and interned at the University Hospital.

Upon his arrival in the United States in 1923, he accepted a teaching post at Georgetown University School of Medicine and taught there until 1932. He also conducted a private practice before joining the medical staff of Soldiers Home in 1955.

Dr. Goldenberg was elected to Active membership in the D. C. Medical Society in 1924 and became a Life member in 1964. He was a member of the American Medical Association and the D. C. Society of Internal Medicine.

He leaves his wife, Mrs. Emily Goldenberg, and 3 daughters, Mrs. Eva Brosseau, of Fontana, Calif.; Mrs. Elyse Dworkin, of Long Beach, Calif.; and Mrs. Jaqueline Pomeranz, of Cleveland, Ohio.

Grover Bache Gill, M.D.

(1890–1968)

Dr. G. Bache Gill, otolaryngologist and sportsman in the Washington area for more than 50 years, died, August 10, 1968 in Ocean City, Md., where he maintained a summer home. He resided at 5800 Deal Place, Chevy Chase, Md.

Dr. Gill was born in Avalon, Va., January 22, 1890. He attended Baltimore City College and graduated in medicine from the Medical College of Virginia, Richmond, in 1916. After interning at Memorial Hospital in Richmond, he went to New York Postgraduate Hospital and the New York Eye and Ear Hospital for training in otolaryngology. He also studied abroad at the University of Vienna in Austria.

He practiced for 14 months on Tangier Island, Va., before entering service in the Medical Corps of the U. S. Army during World War I. He served for 22 months.

Dr. Gill began his practice in the District of Columbia in 1919. For many years he maintained offices at 1726 Eye Street, N.W.

Elected to Active membership in the Medical Society of the District of Columbia in 1920, he transferred to Associate membership in 1953 when he moved his office to his home in Chevy Chase, Md. He was a member of the American Medical Association and the American Academy of Otolaryngology.

Active in sports circles, he was a past commander for 3 terms of the Corinthian Yacht Club, a founder of the President's Cup Regatta, a founder and director of Rosecroft Raceway, and a former director of the U. S. Trotting Horse Association. He was a 32nd degree Mason and a Shriner.

Besides his wife, Mrs. Onda T. Gill, he leaves a son, Grover B. Gill, Jr., of Bethesda, Md., and a sister, Mrs. Raymond K. Foxwell, of Alexandria, Va.

Reuben Goodman, M.D.

(1904–1968)

Dr. Reuben Goodman, Washington dermatologist, died, August 19, 1968 at the Washington Hospital Center. His offices were at 1746 K Street, N.W., before he retired from practice in 1967. He lived at 4501 Connecticut Avenue, N.W.

Dr. Goodman was born in London, England, August 29, 1904. At an early age he came with his family to Syracuse, New York. He graduated in medicine from Syracuse University in 1930 and interned the following year at the Jewish Hospital in Brooklyn, N.Y. He served as resident physician in pathology and X-ray at the same hospital. He secured his training in dermatology at the New York Postgraduate Medical School and Hospital.

For many years he was Assistant Clinical Professor of Dermatology and Syphilology at George Washington University. He was on the associate staff of Children's Hospital and on the attending staff of the Washington Hospital Center and George Washington and D. C. General Hospitals.

Dr. Goodman was one of the founders of the Hebrew Academy of Washington and served as its Vice President from 1944 to 1946. He was a charter member of Brandeis University.

Dr. Goodman was elected to Active membership in the Medical Society of the District of Columbia in 1937, the year he came to Washington. He was a diplomate of the American Board of Dermatology and a member of the American Medical Association, the Baltimore-Washington Dermatological Society, which he served as President in 1945, the National Society of Multiple Sclerosis, and the Jacobi Medical Society. He was a member of B'Nai Israel Congregation.

He is survived by his wife, Mrs. Ida Pachino Goodman; a son, Dr. David Goodman, of Ann Arbor, Michigan; and 2 stepsons, Bernard R. Wilson, of Silver Spring, Md., and Irvin A. Wilson, of Bethesda, Md. He also leaves 2 brothers, Albert Goodman, of Liverpool, Ohio, and Morris Goodman, of Hyattsville, Md., and 2 sisters, Mrs. Rebecca Kopp, of Syracuse, N.Y., and Mrs. Ann Snidman, of Washington.

Lois Sawyer Wilson, M.D.

(1908–1968)

Dr. Lois S. Wilson, obstetrician, died, September 11, 1968 at the Clinical Center of the National Institutes of Health in Bethesda, Md. She lived at 1627 34th Street, N.W.

A native of Yakima, Washington, Dr. Wilson was born, September 7, 1908. She received both her baccalaureate and medical degrees from Stanford University, the latter in 1943. She interned at San Francisco City and County Hospital for a year and spent 2 years as a resident physician at Children's Hospital in San Francisco.

Dr. Wilson, who limited her practice to obstetrics and gynecology, practiced in Berkeley, California, before coming to the District of Columbia in 1952 to join the medical staff of Group Health Association.

She was elected to Active membership in the Medical Society of the District of Columbia in 1953. She was a member of the American Medical Association, the Women's Medical Society of the District of Columbia, and the Washington Gynecological Society.

Surviving her is a sister, Mrs. Mary Graham, 2635 Corralitas Drive, Los Angeles, Calif.

Leukemia in Pregnancy—Vitums and Sites

(*Continued from page 593*)

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Contraindications: Infections caused by nonsusceptible organisms; history of penicillin sensitivity.
Warnings: Acute anaphylaxis (may prove fatal unless promptly controlled) is rare but more frequent in patients with previous penicillin sensitivity, bronchial asthma or other allergies. Resuscitative (epinephrine, aminophylline, pressor amines) and supportive (antihistamines, methylprednisolone sodium succinate) drugs should be readily available. Other rare hypersensitivity reactions include nephropathy, hemolytic anemia, leucopenia and thrombocytopenia. In suspected hypersensitivity, evaluation of renal and hematopoietic systems is recommended.
Precautions: In suspected staphylococcal infections, perform proper laboratory studies including sensitivity tests. If overgrowth of nonsusceptible organisms occurs (constant observation is essential), discontinue penicillin and take appropriate measures. Whenever allergic reactions occur, withdraw penicillin unless condition being treated is considered life threatening and amenable only to penicillin. Penicillin may delay or prevent appearance of primary syphilitic lesions. Gonorrhea patients suspected of concurrent syphilis should be tested serologically for at least 3 months. When lesions of primary syphilis are suspected, dark-field examination should precede use of penicillin. Treat beta-hemolytic streptococcal infections with full therapeutic dosage for at least 10 days to prevent rheumatic fever or glomerulonephritis. In staphylococcal infections, perform surgery as indicated.
Adverse Reactions: (Penicillin has significant index of sensitization): Skin rashes, ranging from maculopapular eruptions to exfoliative dermatitis; urticaria; serum sickness-like reactions, including chills, fever, edema, arthralgia and prostration. Severe and often fatal anaphylaxis has been reported (see "Warnings").
Composition: Tablets—125 mg. (200,000 units), 250 mg. (400,000 units), 500 mg. (800,000 units); Liquid—125 mg. (200,000 units) and 250 mg. (400,000 units) per 5 cc.

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NATIONAL & REGIONAL MEETINGS

November 21–23: Medical Society of the District of Columbia. Washington Hilton Hotel, Washington. Address: Mr. Thomas Sadler, Executive Director, 2007 Eye Street, N.W., Washington, D. C. 20006.

November 21–26: American Heart Association. Americana Hotel, Bal Harbour, Fla. Address: Mr. Rome A. Betts, Executive Director, 44 East 23rd Street, New York, N. Y. 10010.

December 1–4: American Medical Association (Clinical Convention). Miami Beach, Fla. Address: Dr. Ernest B. Howard, Acting Executive Vice President, 535 North Dearborn Street, Chicago, Ill. 60610.

December 4–7: American Medical Women's Association (Annual Meeting). Statler-Hilton Hotel, Boston. Address: Miss Gertrude F. Conroy, Executive Director, 1740 Broadway, New York, N. Y. 10019.

December 5–8: American Rheumatism Association (Interim Meeting). Regency-Hyatt House Hotel, Atlanta. Address: Miss Margaret M. Walsh, Executive Secretary, 1212 Avenue of the Americas, New York, N. Y. 10036.

December 6–7: American Federation for Clinical Research (Eastern Annual Meeting). Somerset Hotel, Boston. Address: Dr. Herbert J. Levine, Chairman, New England Medical Center Hospitals, 171 Harrison Avenue, Boston, Mass. 02111.

December 7–12: American Academy of Dermatology. Palmer House, Chicago. Address: Dr. R. A. Pommerening, Secretary-Treasurer, 911 Seneca Street, Seattle, Wash. 98101.

December 9–11: Southern Surgical Association. Boca Raton Hotel and Club, Boca Raton, Fla. Address: Dr. George H. Yeager, Executive Secretary, 314 Medical Arts Bldg., Baltimore, Md. 21202.

January 18–23: American Academy of Orthopaedic Surgeons. Americana Hotel, New York. Address: Mr. John K. Hart, Executive Secretary, 29 East Madison Avenue, Chicago, Ill. 60602.

January 23–25: Southern Society for Clinical Investigation. Jung Hotel, New Orleans. Address: Mr. James E. Bryan, 2011 Eye Street, N.W., Washington, D. C. 20006.

February 8–13: International Academy of Proctology (Annual Congress and Teaching Seminar). Hollywood Beach Hotel, Hollywood, Fla. Address: Dr. Alfred J. Cantor, Executive Secretary, 147–41 Sanford Avenue, Flushing, N. Y. 11355.

February 18–22: American College of Radiology. Regency-Hyatt House Hotel, Atlanta, Ga. Address: Mr. William C. Stronach, Executive Director, 20 North Wacker Drive, Chicago, Ill. 60606.

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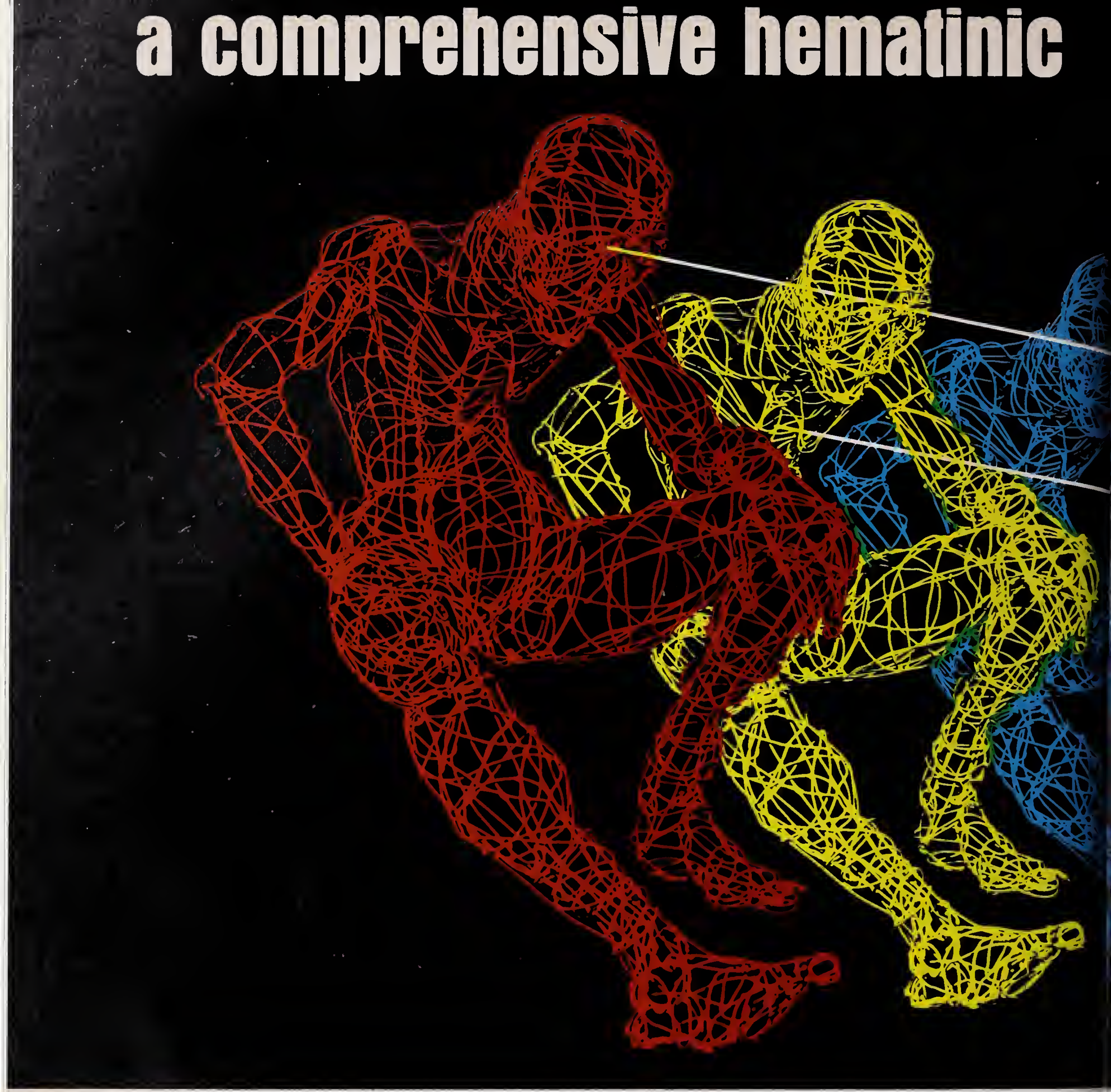

Description: Each Pulvule® contains—

Special Liver-Stomach Concentrate, Lilly (containing Intrinsic Factor)	150 mg.
Cobalamin Concentrate, N.F., equivalent to Cobalamin (The total vitamin B_{12} activity in the Special Liver-Stomach Concentrate, Lilly, and the Cobalamin Concentrate, N.F., is 15 micrograms.)	7.5 mcg.
Iron, Elemental (as Ferrous Fumarate)	110 mg.
Ascorbic Acid (Vitamin C)	75 mg.
Folic Acid	1 mg.

Indications: Trinsicon® (hematinic concentrate with intrinsic factor, Lilly) is a multifactor preparation effective in the treatment of anemias that respond to oral hematinics, including pernicious anemia and other megaloblastic anemias and also iron-deficiency anemia.

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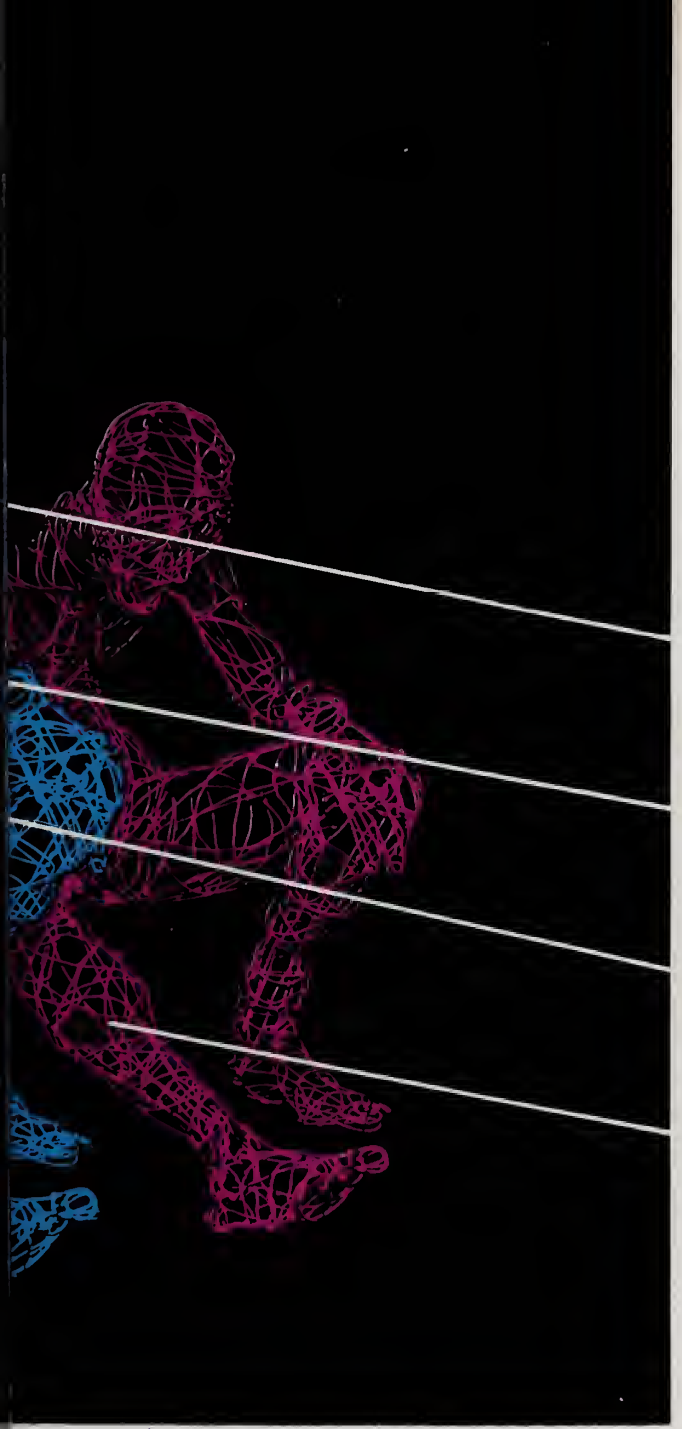

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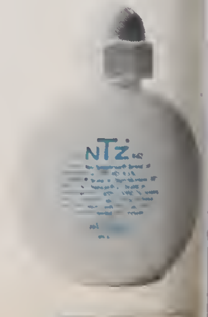

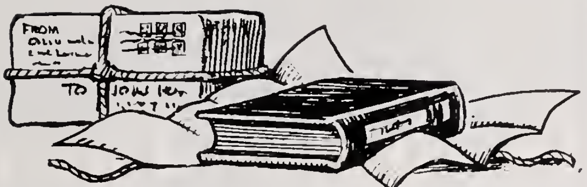

Books Received

The Lung and Its Disorders in the Newborn Infant, 2nd ed. (Volume I in the Series, Major Problems in Clinical Pediatrics). Mary Ellen Avery, M.D.; Alexander J. Schaffer, M.D., Consulting Editor. Price, $9.50. Pp. 285, with 91 illustrations. Philadelphia: Saunders, 1968.

Today's Health Guide: A Manual of Health Information & Guidance for the American Family, revised edition. Edited by W. W. Bauer, M.D. Price, $5.00. Pp. 624, with color transparencies of the organs and systems of the human body. Chicago: American Medical Association, 1968.

Textbook of Otolaryngology, 3rd ed. David D. DeWeese, M.D., and William H. Saunders, M.D. Price, $11.50. Pp. 457, with 396 illustrations. St. Louis: Mosby, 1968.

The Care of the Rheumatoid Hand, 2nd ed. Adrian E. Flatt, M.D., F.R.C.S., F.A.C.S. Price, $14.00. Pp. 234, with 112 illustrations. St. Louis: Mosby, 1968.

Medical Pharmacology: Principles and Concepts, 4th ed. Andres Goth, M.D. Price, $13.50. Pp. 749. St. Louis: Mosby, 1968.

Strabismus in Childhood. Herbert M. Katzin, M.D., and Geraldine Wilson, R.N. Price (paper), $3.95. Pp. 84, with 24 illustrations. St. Louis: Mosby, 1968.

An Atlas of Pelvic Operations, 2nd ed. Langdon Parsons, M.D., and Howard Ulfelder, M.D. Illustrated by Mildred B. Codding, A.B., M.A. Price, $24.00. Pp. 448. Philadelphia: Saunders, 1968.

Internal Medicine—Based on Mechanisms of Disease. Edited by Peter J. Talso, M.D., F.A.C.P., F.A.C.C., and Alexander P. Remenchik, M.D., F.A.C.P.; 31 contributors. Price, $17.50. Pp. 797, with 164 figures. St. Louis: Mosby, 1968.

Growth of the Nervous System: A Ciba Foundation Symposium. Edited by G. E. W. Wolstenholme and Maeve O'Connor. Price, $12.00. Pp. 295, with 101 illustrations. Boston: Little, 1968.

Systemic Mycoses: A Ciba Foundation Symposium in commemoration of William Balfour Baikie. Edited by G. E. W. Wolstenholme and Ruth Porter. Price, $12.00. Pp. 287, with 49 illustrations. Boston: Little, 1968.

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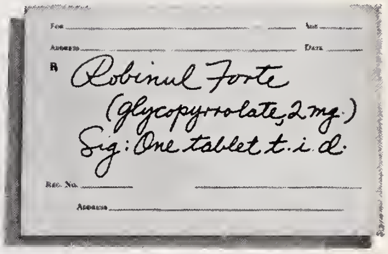
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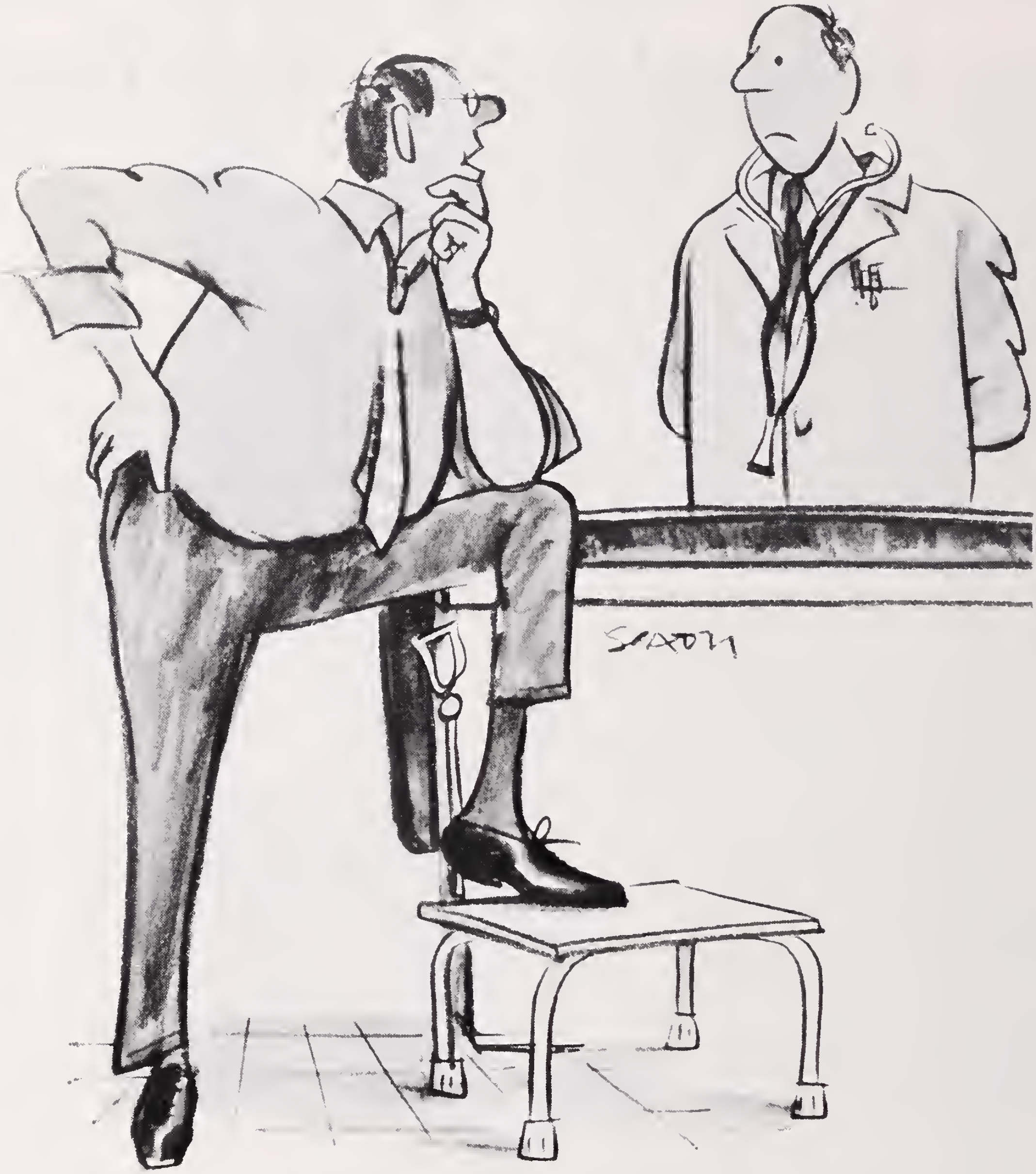

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Book Reviews

Textbook of Otolaryngology, 3rd ed. David D. DeWeese, M.D., and William H. Saunders, M.D. Price, $11.50. Pp. 457, with 396 illustrations. St. Louis: Mosby, 1968.

The third edition of this text remains a definitive work of its kind. Originally the authors wrote for an audience of medical students and general practitioners, but any physician who ventures into the ENT regions should profit from its use.

The format of this edition remains very similar to prior ones. The main changes lie in updating of knowledge in this rapidly changing field. The organization of material is concise and generally comprehensive. New techniques of middle ear surgery have been added as well as advances in laryngeal work. The illustrations are, in general, instructive and appropriately spaced.

I believe this text achieves its goal—a general survey of the field of otolaryngology.

Robert E. Pumphrey, M.D.

The First International Congress of Orthoptists. Transactions of a Congress held at the Royal College of Surgeons of England, July 3, 4, 5, 1967. Price, $14.25. Pp. 376. St. Louis: Mosby, 1968.

The Congress transctions are divided into 6 sections, each followed by a panel discussion. The papers are presented by orthoptists and ophthalmologists. Most of the papers are illustrated by black and white photos, graphs, and diagrams.

Anomalies of Binocular Vision and Orthoptic Therapy are discussed in the first 2 sections. In general, there is little new information here; most articles stress the role of orthoptic evaluation and treatment. For the ophthalmologist who has fallen out of the habit of referring patients to the orthoptist these sections will be of some interest.

The papers on surgery related to ocular motility, all by ophthalmologists, are well written and clearly illustrated. Of particular interest are the following: "Treatment of A and V Syndromes," "Plastic Materials in Muscle Surgery," "Blowout Fractures of the Orbit," and "Traumatic Anomalies of Binocular Vision."

One section contains a variety of papers primarily of interest to the orthoptist. This is followed by a section on Education of Orthoptists. Finally, a section on Miotics ends the symposium.

Complete author and subject indices are included.

There is no paper on pleoptics.

The general quality of the papers is excellent. The authors are knowledgeable and informative. In spite of the international distribution of authors the organization of the symposium is good. There are a few areas where terms are not well defined, particularly when naming orthoptists' examining instruments. Here a glossary would be helpful.

I would urge all orthoptists to read this book and study it.

Ophthalmologists should be aware of the information it contains and refer to it as an authoritative source of information regarding some phases of ocular motility. Unfortunately, this book is not adequate to serve as a textbook of ocular motility, which is so badly needed.

Henry J. Starr, M.D.

Dyslexia: Diagnosis and Treatment of Reading Disorders. Edited by Arthur H. Keeney, M.D., D.Sc., and Virginia T. Keeney, M.D. Price, $12.00. Pp. 182, with 24 illustrations. St. Louis: Mosby, 1968.

The diagnosis and treatment of reading disorders has become an increasingly more frequent and perplexing problem for those physicians concerned with the welfare and guidance of pediatric patients.

This volume, directed primarily toward ophthalmic and medical practitioners, represents an extensive review of all aspects of dyslexia, or reading retardation. Contributors are from the fields of psychiatry, neurology, ophthalmology, psychology, audiology, education, and the speech and reading disciplines.

Dyslexia is described as a basic deficiency of visual and auditory symbolization primarily affecting the language areas of reading, spelling and writing, and frequently the arithmetic functions. Several classifications of the disorders are offered (some conflicting) and detailed discussions pursue the causative roles of heredity, neurologic dysfunction, visual and oculomotor imbalances, social and cultural deprivation, inadequate teaching techniques, speech and hearing deficits, and numerous other factors.

Considerable emphasis is placed on the early recognition of such disorders, preferably before entering first grade, and the guidelines for diagnostic evaluation promote the concept of interdisciplinary facilities, utilizing specialists in pediatrics, clinical psychology, neurology, vision, hearing, speech, and special education.

In addition to correcting vision and hearing deficits and stabilizing any emotional disorders, the most

Book Reviews (*Continued*)

reasonable and effective treatment measures revolve around special education programs with low pupil-teacher ratio, classroom aids, and individually tailored instruction for each child. One entire chapter devoted to hand-eye dominance patterns advocates eye occlusion when mixed lateralization is present. There is little to support this theory from other contributors or from general experience in ophthalmology, except in rare cases.

Although many questions remain unanswered regarding the specific etiology of dyslexia, the comparative effectiveness of different treatment methods, and where the primary responsibility for management lies, this text offers an extensive review of current thinking by acknowledged experts and should be a valuable source book for anyone interested in this field.

John F. O'Neill, M.D.

Medical Physiology, 12th ed. Volumes I and II. Edited by Vernon B. Mountcastle, M.D. Price per 2-volume set, $24.00. Pp. 1054 (Vol. I.), 1055–1858 (Vol. II), with 880 illustrations, including 1 in color. St. Louis: Mosby, 1968.

The new edition of Medical Physiology has been extensively revised with the addition of several chapters and major revisions of most of the remaining ones.

As in any text of this type which is prepared by numerous contributors, much variability in both form and content of the individual sections is found. One entire volume is devoted to neurophysiology, and this subject is probably covered in more detail than in any current work of this type. Certain other subjects receive coverage that is less detailed and hardly current. A major deficit is to be found in the section on the physiology of absorption. Major advances that have occurred in the understanding of the role of bile salts, micelle formation, and intracellular events in lipid transport are not even mentioned. These and several minor inadequacies detract somewhat from the overall excellence of this publication.

Many features require favorable comment. The usual approach in the presentation, providing the experimental background of the material discussed, a refreshing contrast to the authoritative statement of "facts" seen in many texts, is one that will appeal to the inquiring mind. In general, consideration is also given to the historical background of a given area. For those who wish to survey an area in greater depth, an extensive bibliography is provided.

The failure to apply physiology to clinical disorders may make the text less attractive to the average medical student or practicing physician. The work can be recommended to the student of physiology and particularly to those with an interest in neurophysiology.

Martin G. Durkin, M.D.

Proceedings of the Second International Symposium on Plastic and Reconstructive Surgery of the Eye and Adnexa. Byron Smith, M.D., and John Marquis Converse, M.D., Co-chairmen; Donald Wood-Smith, F.R.C.S.E., and Margaret F. Obear, M.D., Secretaries; and 60 contributors. Price, $31.00. Pp. 548, with 303 illustrations. St. Louis: Mosby, 1967.

Edited by Co-chairmen of the Symposium, Drs. Byron Smith and John Marquis Converse, each of whom is outstanding in the fields of ophthalmology and plastic and reconstructive surgery, respectively, this volume lists among its contributors members of both specialties who have made this overlapping segment of ophthalmic and plastic surgery a significant part of their practices.

There are 63 chapters or subdivisions. The first of these covers the underlying principles governing this phase of these 2 disciplines. Subsequent chapters cover the more commonly encountered benign and malignant processes involving the lids and adnexa, as well as the early and late care of traumatic defects. Five chapters deal with cosmetic surgery of the lids, including a panel discussion of this topic.

Based on a symposium, the book incorporates several panel discussions on the more recent, and possibly controversial, techniques, such as the use of foreign implants and injections, thus enabling the reader to better evaluate their merits and the possible danger inherent in their application.

This volume would benefit those who are developing skills in this field. It can also be recommended as a reference for those actively engaged in the practice of plastic and ophthalmic surgery and as a source of current information on this rapidly advancing phase of surgery.

Albert F. Fleury, M.D.

Internal Medicine—Based on Mechanisms of Disease. Edited by Peter J. Talso, M.D., F.A.C.P., F.A.C.C., and Alexander P. Remenchik, M.D., F.A.C.P.; with 31 contributors. Price, $17.50. Pp. 797, with 164 figures. St. Louis: Mosby, 1968.

The authors state that their purpose in writing this book is to provide the student with a portable, ready reference to the problems and conditions he will find during his early medical training.

The book presents a rather good section on Basic Mechanisms of Disease with a very lucid subsection on the genetic factors in disease. Also well done is the chapter on immunity and host defense. From there, the book is a rather standard review, in a very basic way, of the common disease states arranged by organ system. I found the list of references to be somewhat lacking.

The authors seem to achieve their purpose in setting forth a lightweight (797 pages) introduction to medical problems. It is quite well written but adds little to the better known, more complete textbooks of internal

(*Continued on page 50*)

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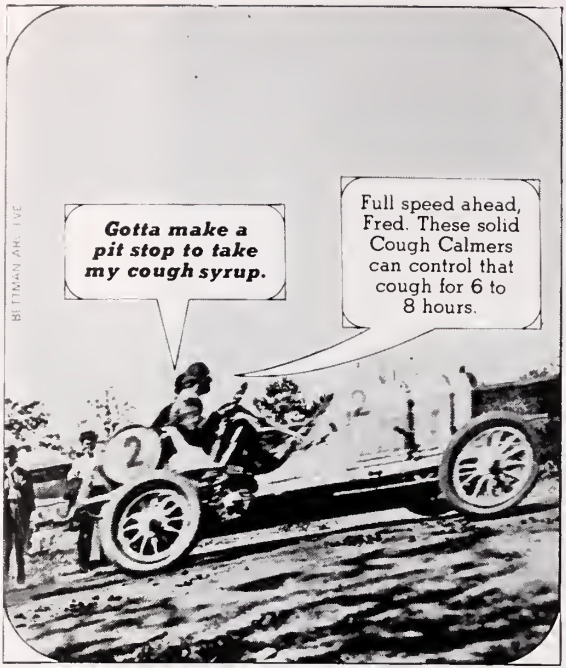
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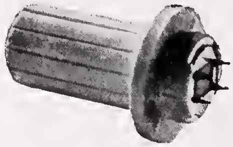

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Book Reviews (*Continued*)

medicine. It also has the disadvantage of providing the student with superficial knowledge about disease states, thus discouraging him from a more thorough literature search. Its audience would seem no wider than junior and senior medical students.

JOSEPH P. SWIFT, M.D.

Ciba Foundation Study Group No. 31: Nutrition and Infection. Edited by G. E. W. WOLSTENHOLME AND MAEVE O'CONNOR. Price, $3.50. Pp. 144, with 20 illustrations. Boston: Little, 1968.

This small volume, the transactions of a 1-day symposium attended by an elite study group of 16 individuals, encompasses many of the significant features regarding interactions between infection and nutrition. The most striking aspects of malnutrition are found in the developing nations of the world as well as the most overwhelming infections. Thus, opportunities for medical research accomplishment are fantastic. The participants in this symposium comprise highly able physicians and investigators of clinical problems surrounding malnutrition.

It may be of more than passing interest to mention that only 1 American (Dr. Paul Gyorgy) was a member of this Study Group. Perhaps it points up an important aspect of international medical research, namely, that the United States has not contributed to this field in proportion to its skills and resources. Is it because medical opportunities are so great in this country that competent clinical investigators are loath to spend long enough periods abroad to make significant accomplishments? This sociological factor, coupled with the rapidly disappearing sources of funds for foreign research, will surely leave us far behind in the rewarding field of international health. This can only be our loss, because the opportunities to learn are vast and the chances very great to help the unfortunate masses in the have-not countries of the world, not only through well-directed preventive and curative medicine but by adding to knowledge through research.

Like many of the Ciba Foundation Study Group reports this one is authoritative, lucid and readable. Each paper is followed by much transcribed discussion which makes the topic come alive. Nutrition as a medical discipline in the American medical environment is difficult to define. This is hardly the case in truly poverty areas where the problems of malnutrition stand out in glaring prominence. Yet in underdeveloped countries the almost universal lack of accurate public health statistics and often insuperable difficulties of controlling experimental situations where largely illiterate and often superstitious patients predominate make it difficult to obtain precise information. As one of the participants, Dr. Maegraith, puts it, "People who live in sophisticated countries seldom realize the difficulties of tackling [these problems] in the field."

Nevertheless, one is struck by the amount of knowledge which has been acquired when one reads this little

Book Reviews *(Continued)*

volume. Placental malaria as a cause of fetal growth failure is an admirably controlled study from Uganda. Documentation of the clinical features, nature of the anemia, and treatment of protein deficiency (kwashiorkor) is becoming more precise. Studies of the effect of infection on intestinal absorption and of the incidence of lactase deficiency are impressive. Discovery of the protective action of hemoglobin S and of glucose-6-phosphate dehydrogenase deficiency on the incidence and course of *P. falciparum* malaria is a landmark of recent achievement in tropical medicine.

For the practicing physician this book is highly recommended because it is concise and for its size is quite comprehensive. Although, paradoxically, there is more knowledge of nutritional states in underdeveloped countries than in our own, soon this situation promises to change when careful surveys now beginning in a number of states are completed. Kwashiorkor has been recognized in Washington, D.C. Doubtless many of the malnutrition syndromes thought to be nonexistent in the United States are going to be found when they are looked for, especially in poverty areas. For the thoughtful physician who wishes to become more familiar with some of these problems this volume will be most helpful.

James A. Halsted, M.D.

Systemic Mycoses: A Ciba Foundation Symposium in commemoration of William Balfour Baikie. Edited by G. E. W. Wolstenholme and Ruth Porter. Price, $12.00. Pp. 287, with 49 illustrations. Boston: Little, 1968.

In keeping with previous Ciba Foundation Symposia, Systemic Mycoses represents the thoughts and experience of many internationally prominent workers in the field of mycology. While the meeting was held in Nigeria and much of the stress was placed upon the occurrence of fungal disease in Africa, there is also extensive reference to mycotic diseases throughout the rest of the world.

The Symposium is divided into 4 sections: Pathology, Clinical Aspects, Epidemiology, and Therapy. Presentations are followed by discussion and questions among the experts. Excellent bibliographies follow each section, although many of the referenced journals are not generally available in the United States.

Clinical correlation adds spice to the basic science portions of the meeting, while basic biologic and chemical support is offered for the clinical and therapeutic discussions.

Because the subject of systemic mycoses is of limited interest to most physicians, these proceedings will not be widely read. However, for those of us who deal with this subject as part of our everyday practice, this book contains a wealth of information not readily found elsewhere.

Richard L. Levin, M.D.

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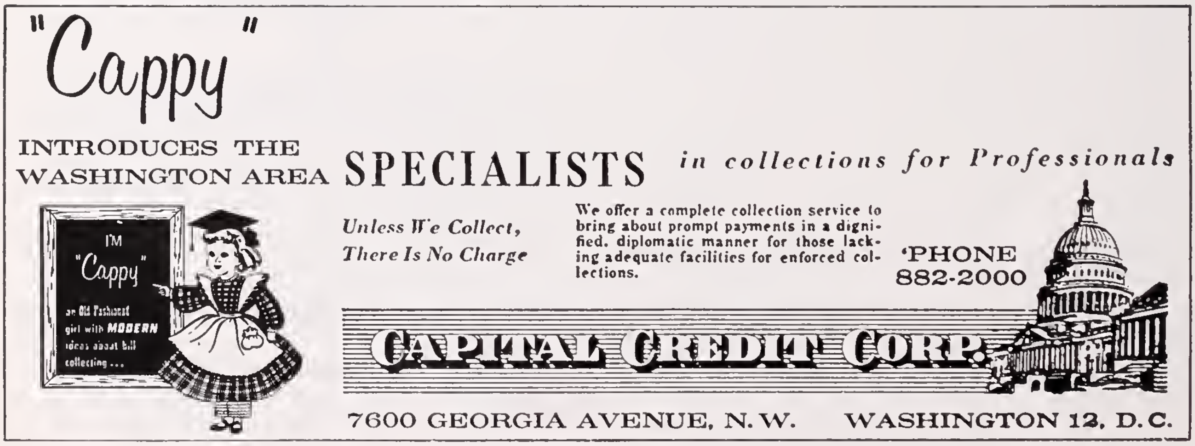
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T-text

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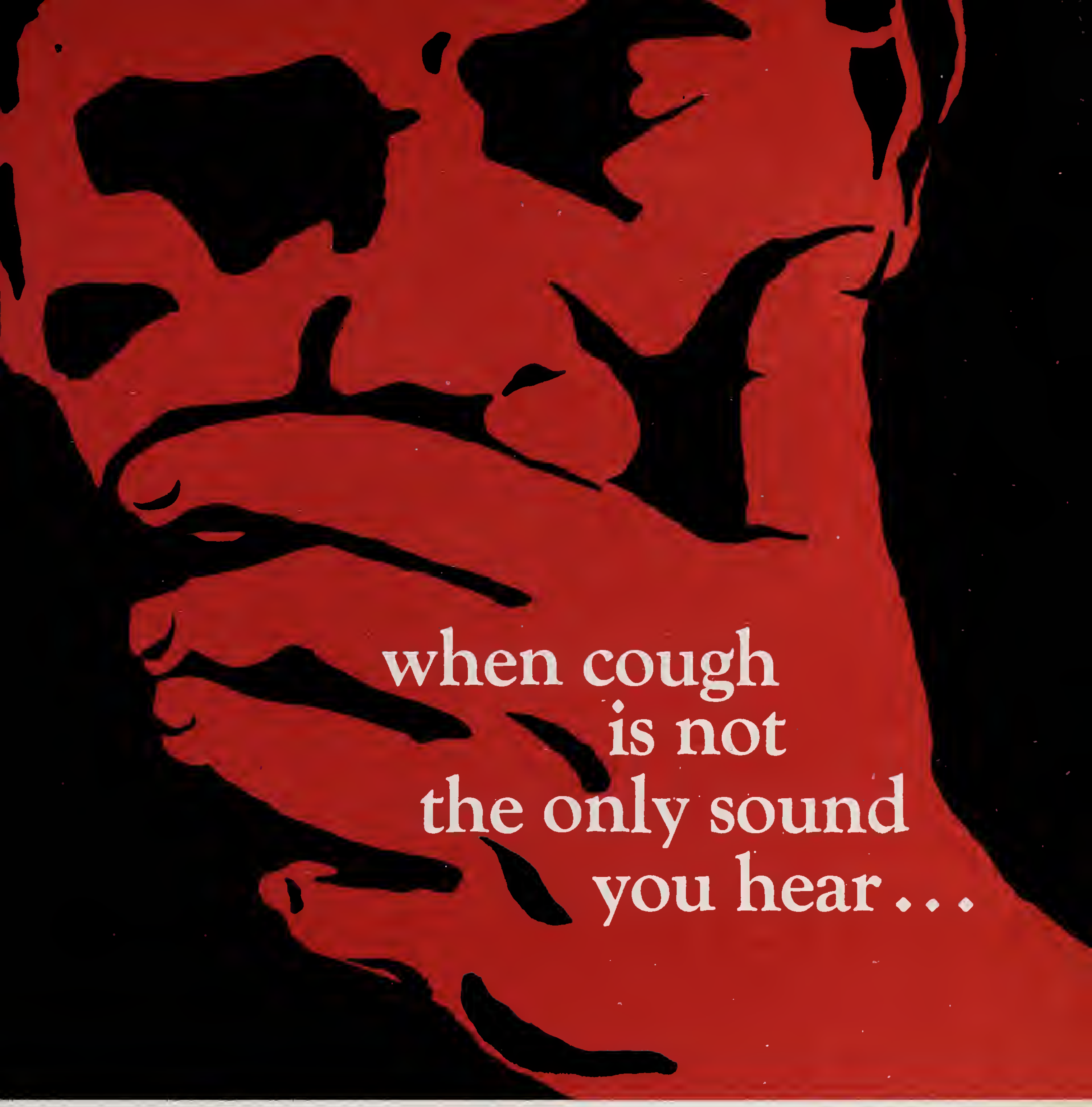

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Warning: In renal impairment, since liver toxicity is possible, lower doses are indicated; during prolonged therapy consider serum level determinations. Photodynamic reaction to sunlight may occur in hypersensitive persons. Photosensitive individuals should avoid exposure; discontinue treatment if skin discomfort occurs.

Precautions: Drowsiness, anorexia, slight gastric distress can occur. In excessive drowsiness, consider longer dosage intervals. Persons on full dosage should not operate vehicles. Nonsusceptible organisms may overgrow; treat superinfection appropriately. Treat beta-hemolytic streptococcal infections at least 10 days to help prevent rheumatic fever or acute glomerulonephritis. Tetracycline may form a stable calcium complex in bone-forming tissue and may cause dental staining during tooth development (last half of pregnancy, neonatal period, infancy, early childhood).

Adverse Reactions: *Gastrointestinal*—anorexia, nausea, vomiting, diarrhea, stomatitis, glossitis, enterocolitis, pruritus ani. *Skin*—maculopapular and erythematous rashes; exfoliative dermatitis; photosensitivity; onycholysis, nail discoloration. *Kidney*—dose-related rise in BUN. *Hypersensitivity* reactions—urticaria, angioneurotic edema, anaphylaxis. *Intracranial*—bulging fontanels in young infants. *Teeth*—yellow-brown staining; enamel hypoplasia. *Blood*—anemia, thrombocytopenic purpura, neutropenia, eosinophilia. *Liver*—cholestasis at high dosage.

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Precautions: In the elderly and debilitated, and in children over six, limit to smallest effective dosage (initially 10 mg or less per day) to preclude ataxia or oversedation, increasing gradually as needed and tolerated. Not recommended in children under six. Though generally not recommended, if combination therapy with other psychotropics seems indicated, carefully consider individual pharmacologic effects, particularly in use of potentiating drugs such as MAO inhibitors and phenothiazines. Observe usual precautions in presence of impaired renal or hepatic function. Paradoxical reactions (*e.g.*, excitement, stimulation and acute rage) have been reported in psychiatric patients and hyperactive aggressive children. Employ usual precautions in treatment of anxiety states with evidence of impending depression; suicidal tendencies may be present and protective measures necessary. Variable effects on blood coagulation have been reported very rarely in patients receiving the drug and oral anticoagulants; causal relationship has not been established clinically.

Adverse Reactions: Drowsiness, ataxia and confusion may occur, especially in the elderly and debilitated. These are reversible in most instances by proper dosage adjustment, but are also occasionally observed at the lower dosage ranges. In a few instances syncope has been reported. Also encountered are isolated instances of skin eruptions, edema, minor menstrual irregularities, nausea and constipation, extrapyramidal symptoms, increased and decreased libido—all infrequent and generally controlled with dosage reduction; changes in EEG patterns (low-voltage fast activity) may appear during and after treatment; blood dyscrasias (including agranulocytosis), jaundice and hepatic dysfunction have been reported occasionally, making periodic blood counts and liver function tests advisable during protracted therapy.

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Corrected Transposition of Great Vessels

M. S. Valiathan, M.B.B.S., L. W. Perry, M.D., and L. P. Scott III, M.D.

Radiosensitivity of Head and Neck Cancer

Domenic E. Sabatini, M.D.

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Streptococci and Virus Infections

James G. Cumming, M.D.

Inappropriate Lactation

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Transvenous Standby Pacemaking

N. P. D. Smyth, M.D., J. M. Bacos, M.D., and M. R. Boivin, C.P.T.

Cardiac Pacing in Myocardial Infarction

Philip Samet, M.D.

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DOSAGE: **INJECTABLE** — Average adult dose: one ampul, 2 cc. (60 mg. orphenadrine citrate) I.M. or I.V. May be repeated every 12 hours. Relief may be maintained with one NORFLEX tablet b.i.d. **TABLETS** — Two tablets per day for adults, one in the morning, one in the evening. Each tablet contains 100 mg. orphenadrine citrate.

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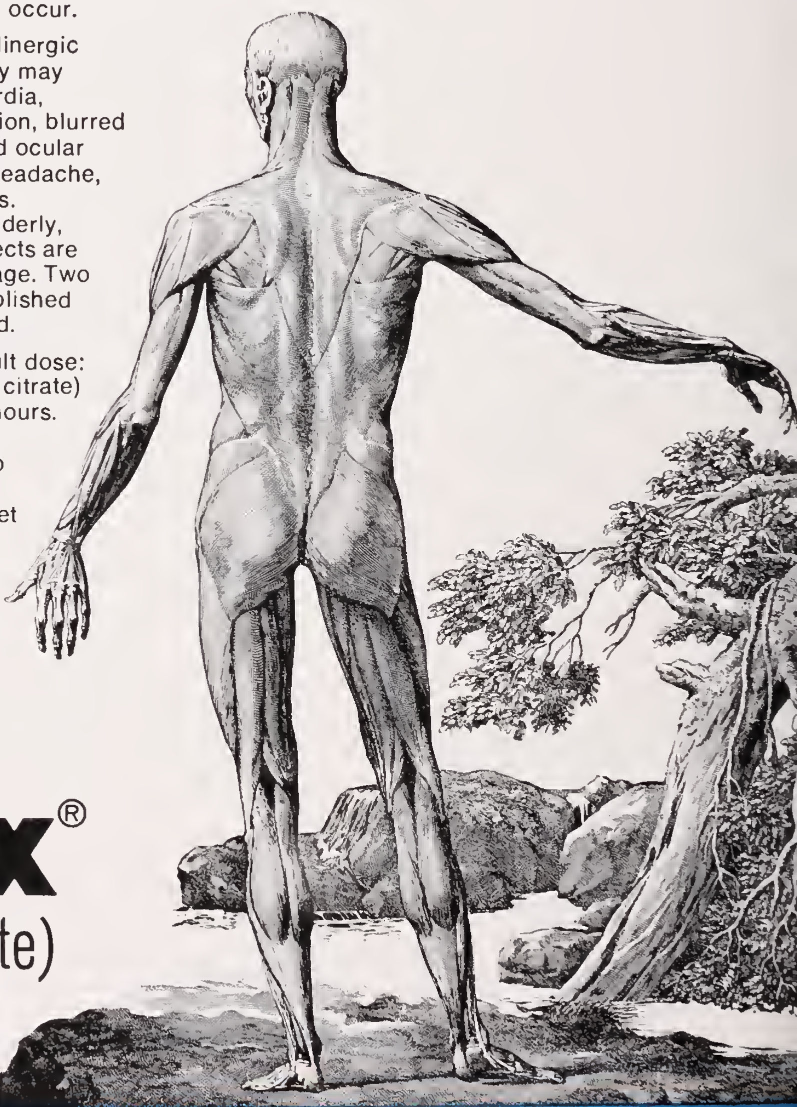

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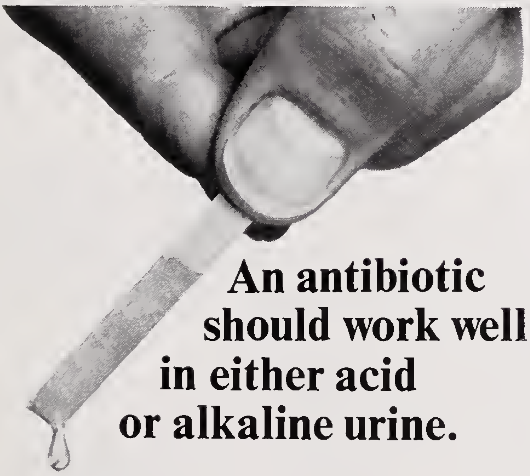

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Contraindication: History of hypersensitivity to demethylchlortetracycline.

Warning: In renal impairment, usual doses may lead to excessive accumulation and liver toxicity. Under such conditions, lower than usual doses are indicated, and, if therapy is prolonged, serum level determinations may be advisable. A photodynamic reaction to natural or artificial sunlight has been observed. Small amounts of drug and short exposure may produce an exaggerated sunburn reaction which may range from erythema to severe skin manifestations. In a smaller proportion, photoallergic reactions have been reported. Patients should avoid direct exposure to sunlight and discontinue drug at the first evidence of skin discomfort. Necessary subsequent courses of treatment with tetracyclines should be carefully observed.

Precautions: Overgrowth of nonsusceptible organisms may occur. Constant observation is essential. If new infections appear, appropriate measures should be taken. In infants, increased intracranial pressure with bulging fontanels has been observed. All signs and symptoms have disappeared rapidly upon cessation of treatment.

Side Effects: Gastrointestinal system – anorexia, nausea, vomiting, diarrhea, stomatitis, glossitis, enterocolitis, pruritus ani. Skin – maculopapular and erythematous rashes; a rare case of exfoliative dermatitis has been reported. Photosensitivity; onycholysis and discoloration of the nails (rare). Kidney – rise in BUN, apparently dose-related. Transient increase in urinary output, sometimes accompanied by thirst (rare). Hypersensitivity reactions–urticaria, angioneurotic edema, anaphylaxis. Teeth – dental staining (yellow-brown) in children of mothers given this drug during the latter half of pregnancy, and in children given the drug during the neonatal period, infancy and early childhood. Enamel hypoplasia has been seen in a few children. If adverse reaction or idiosyncrasy occurs, discontinue medication and institute appropriate therapy. Demethylchlortetracycline may form a stable calcium complex in any bone-forming tissue with no serious harmful effects reported thus far in humans.

Average Adult Daily Dosage: 150 mg q.i.d. or 300 mg b.i.d. Should be given 1 hour before or 2 hours after meals, since absorption is impaired by the concomitant administration of high calcium content drugs, foods and some dairy products. Treatment of streptococcal infections should continue for 10 days, even though symptoms have subsided.

Capsules: 150 mg; Tablets: film coated – 300 mg, 150 mg and 75 mg of demethylchlortetracycline HCl.

355-8

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Dosage: Should be adjusted according to individual patient response. Average and maximum recommended dose is 1 tablet three times a day; in the a.m., early p.m., and at bedtime. See product literature for full prescribing information. Supply: Robinul (glycopyrrolate 1 mg.): Robinul Forte (glycopyrrolate 2 mg.): Robinul-PH (glycopyrrolate 1 mg.) with phenobarbital 16.2 mg. (Warning: may be habit forming); Robinul-PH Forte (glycopyrrolate 2 mg.) with phenobarbital 16.2 mg. (Warning: may be habit forming), in bottles of 100 and 500 tablets. A. H. Robins Company, Richmond, Va. 23220.

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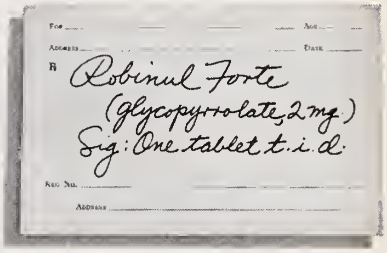

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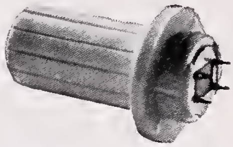

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Book Reviews

Strabismus Ophthalmic Symposium (I). Edited by James H. Allen, M.D. Price, $23.00 Pp. 476, with 181 illustrations. St. Louis: Mosby, 1950; second printing, 1968.

This volume is a second printing, with variations, of one printed in the year 1950. It was the first of this type of Ophthalmic Symposia held in the United States. The Symposium explores with the reader the possible causes of strabismus and its treatment, surgical and nonsurgical. It is recommended for complete study by the ophthalmic specialist and the orthoptician. Eleven eminent authors have contributed individual chapters to this outstanding book. Most thoroughly discussed in the chapters are the anatomy, physiology, and surgical procedures on the extraocular muscles, voluntary and involuntary mechanisms, congenital anomalies, and the use of drugs and prisms in the treatment of ocular deviations.

However, considering all the knowledge we have accumulated throughout the years regarding the subject of strabismus we still have not reached a "rule of thumb" on the surgical procedure in its treatment. As one of the contributing editors, Dr. C. S. O'Brien, so aptly puts it in his chapter on Surgical Management of Convergent Strabismus (referring to surgery on the extraocular muscles): "The mathematical millenium is not in sight and it is doubtful if it ever will be."

No one as yet can offer a mathematical formula in advising how many extraocular muscles should be operated on at one time, and how much a muscle should be lengthened or shortened in each case. Because this is true at the present time, the same author wisely advises conservatism in the surgical approach to the problem of strabismus.

Thomas A. Egan, M.D.

The Lung and Its Disorders in the Newborn Infant, 2nd ed. (Volume I in the Series, Major Problems in Clinical Pediatrics). Mary Ellen Avery, M.D.; Alexander J. Schaffer, M.D., Consulting Editor. Price, $9.50. Pp. 285, with 91 illustrations. Philadelphia: Saunders, 1968.

The original edition of this monograph, published in 1964, brought together for the first time in one volume the essence of current knowledge about pulmonary physiology and disease in the newborn. Since then this book truly has become a classic. Its broad appeal may be attributed to its unique presentation of an important area of medicine for the newborn on a foundation of basic physiology.

Publication of this second edition was prompted by ever-increasing data as well as some inadvertent omissions in the previous edition. Much of the text has not been substantially rewritten, but new material has been added in all the major topics. The one entirely new chapter, Roentgenographic Evaluation of the Chest, by Dr. John P. Dorst, deals at some length with radiographic technique, which seems of limited value to the pediatrician. However, the pitfalls of radiographic interpretation pointed out are quite helpful.

As before, the heart of the book is the chapter on Hyaline Membrane Disease. This is one of the more comprehensive reviews available on this major unsolved clinical problem. The dilemma facing the clinician treating this disorder is well illustrated by the paucity of firmly established therapeutic modalities in the face of an overwhelming amount of clinical and laboratory data. During the 4 years separating the 2 editions, only respirator therapy could claim recognition as new and promising treatment. Enthusiasm for this, however, is tempered by accompanying reports of the pulmonary complications of such therapy and lack of success in babies under 1,500 grams.

A final section is devoted to artificial respiration. Principles, as well as technique, are clearly presented. Clinicians will find the pointers on resuscitation of the newborn particularly helpful. In the rapidly developing area of respirator therapy there is also much valuable material, although the physician who uses respirators for prolonged support of newborns may wish for more detailed technical advice than is offered in this chapter.

The book is concise and highly readable. There is an exhaustive bibliography and the index is adequate. The work promises to fulfill the author's desire to provide a "kind of bridge, over which the clinician and the investigator, whose common aims are sometimes obscured by their different approaches, can meet each other when it seems advantageous."

Ernest N. Kraybill, M.D.

The Care of the Geriatric Patient, 3rd ed. Edited by E. V. Cowdry, Ph.D., Sc.D. (Hon.). Price, $15.75. Pp. 430, with 19 illustrations. St. Louis: Mosby, 1968.

This is the third edition of this pioneering text on the care of the elderly patient. Its 28 revised or entirely new chapters range from considerations of the doctor-patient relationship to geriatrics around the world. These contributions are uneven in value, but taken together they offer a reasonably comprehensive body

(Continued on page 18)

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Book Reviews

(*Continued from page 15*)

of knowledge of this increasingly important field. However, a truly major textbook of geriatric medicine does not yet exist.

Nineteen million Americans are over 65 today, and it is estimated that there will be 33 million retired Americans alive in the year 2000. Yet research in the fields of gerontology and geriatrics is limited, and few medical schools offer major courses in geriatric medicine. This volume should be widely used in teaching.

One value of this book is its emphasis on the care of the older person in the community where 96 per cent of our 65-plus population reside.

Rehabilitation is also given appropriate coverage. Underlying the entire work is a balanced proportion of optimism and realism. It is made clear that much can be done in our clinical care of the elderly patient.

Robert N. Butler, M.D.

Strabismus in Childhood. Herbert M. Katzin, M.D., and Geraldine Wilson, R.N. Price (paper), $3.95. Pp. 84, with 24 illustrations. St. Louis: Mosby, 1968.

This latest edition of "Strabismus in Childhood," by Herbert M. Katzin and Geraldine Wilson, has much to recommend it. Parents who read this short (84-page) paperback book will undoubtedly acquire some knowledge concerning the nature of their child's problem and the therapeutic methods available to attain the universally acknowledged desiderata of good vision in both eyes and straight eyes that work together in a normal manner.

Unfortunately, the work does not remain on the high ground of generally accepted concepts and procedures. Much of the material is unsubstantiated and controversial. This is especially relevant to the authors' repeated assertions that muscle abnormalities are a major cause of strabismus and also to their undue emphasis on the role of orthoptics in patient management. Oculists who do not share such opinions may well refrain from recommending this book to the parents of their strabismic patients.

It would seem that in this area there is a need for a comprehensive, insightful, and simply written digest of currently accepted principles directed to the lay public. It would also seem that the authors have fallen somewhat short of the goal.

David S. Friendly, M.D.

Anesthesia for Infants and Children, 3rd ed. Robert M. Smith, M.D. Price, $17.50. Pp. 530, with 240 illustrations. St. Louis: Mosby, 1968.

The appearance of this third edition in a period of 10 years is correctly explained on the basis of change of concepts in types of drugs administered, the methods of delivery, the importance of multimonitoring, and the increased responsibility of the anesthesiologist for postoperative care, with all its facets. The transition from a text of basic fundamentals to a worthy, mature reference book required the embellishment and development of modern medical knowledge as related to infant anesthesia and surgery not found in the 2 earlier editions. I believe the 31 chapters in this book accomplish this purpose admirably.

The third chapter of the book, Respiratory Physiology in Infants and Children, by Charles D. Cook, M.D. and Etsuro K. Motogama, M.D., with its new material and illustrations is worth the price of the new edition, both to the student and the practicing physician.

The additional 47 pages, over the second edition, supply new and improved illustrations, graphs, tracings, and photography that have immeasurably improved the value of this volume.

The extensive bibliographies make this book a worthy reference for the expert. For the neophyte it is a complete text in pediatric anesthesiology.

This book should be a welcome addition to any medical library.

Charles E. Fierst, M.D.

Growth of the Nervous System: A Ciba Foundation Symposium. Edited by G. E. W. Wolstenholme and Maeve O'Connor. Price, $12.00. Pp. 295. with 101 illustrations. Boston: Little, 1968.

This volume contains a series of papers delivered at a Symposium on Growth of the Nervous System held in June 1967. The contributors are all well recognized experts in their field from diffeient parts of the world. The aspect of neural growth discussed is cell development rather than organ maturation. Specifically, the majority of papers are concerned with the development of nerve processes and the establishment of synaptic contacts.

Individual chapters are generally well written and tend to be a summary of an investigator's research experience over many years. The discussions between the participants are usually lively and allow the reader some insight into the problems of the scientist.

A weakness of the symposium is the general failure to develop a theme. The 4 sessions include: (1) development of specific neuronal connections, (2) development of movement, (3) the role of chemical signals in nervous system development, and (4) trophic interaction. At the end of each session the reader is not left with a formulation of principles related to these subjects but rather with pieces of the whole.

This book is concerned with cell biology and will be a welcome addition to the library of scientists concerned with basic biological processes in the nervous system. It is of limited value to medical students or residents and would offer little or no pertinent information to practicing physicians.

Gerald M. Fenichel, M.D.

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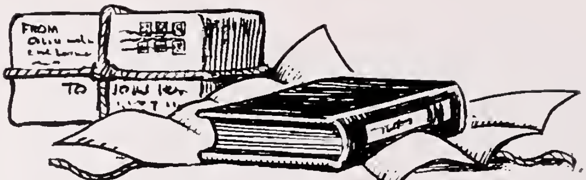

Books Received

Atlas of Precautionary Measures in General Surgery. Ivan D. Baronofsky, M.D., Ph.D. Price, $23.50. Pp. 281, illustrated with 111 plates and 132 figures by Daisy Stilwell. St. Louis: Mosby, 1968.

Current Diagnosis-2. Edited by Howard F. Conn, M.D., and Rex B. Conn, Jr., M.D.; 332 contributors. Price, $21.75. Pp. 990. Philadelphia: Saunders, 1968.

Identification and Individuality: Instincts Fundamental to Human Behavior. Monograph Series. John T. Flynn, M.D. Price, $4.95. Pp. 82. New York: Beekman Press, 1968.

Textbook of Radiologic Technology, 4th ed. Charles A. Jacobi, B.Sc., R.T. (A.R.R.T.), and Don Q. Paris, R.T. (A.R.R.T.). Price, $12.75. Pp. 480, with 384 illustrations. St. Louis: Mosby, 1968.

Synopsis of Surgery. Richard D. Liechty, M.D., and Robert T. Soper, M.D., with 21 contributors. Price, $12.50. Pp. 1091, with 346 illustrations. St. Louis: Mosby, 1968.

Women In Medicine. Published for The Josiah Macy, Jr. Foundation. Carol Lopate. Price, $5.95. Pp. 204. Baltimore: Johns Hopkins Press, 1968.

Cystic Fibrosis. Ciba Foundation Study Group No. 32. Edited by Ruth Porter and Maeve O'Connor. Price, $3.95. Pp. 150, with 36 illustrations. Boston: Little, 1968.

Hearing Mechanisms in Vertebrates. A Ciba Foundation Symposium. Edited by A. V. S. de Reuck and Julie Knight. Price, $12.00. Pp. 320, with 107 illustrations. Boston: Little, 1968.

The Mentally Abnormal Offender. A Ciba Foundation Symposium. Edited by A. V. S. de Reuck and Ruth Porter. Price, $10.00. Pp. 260, with 2 illustrations. Boston: Little, 1968.

Atlas of Fluorescence Fundus Angiography. Shinichi Shikano, M.D., and Koichi Shimizu, M.D. Price, $28.00. Pp. 201, illustrated (some in color). Philadelphia: Saunders, 1968.

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MEDICAL ANNALS
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December 1968
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Corrected Transposition of the Great Vessels

A Review of Five Cases

M. SANKARAN VALIATHAN, M.B.B.S.,* LOWELL W. PERRY, M.D.,† AND LEWIS P. SCOTT III, M.D.‡

SINCE THE ORIGINAL DESCRIPTION in 1875 by vonRokitansky, corrected transposition of the great vessels has been extensively studied.[1-4] Much of the recent interest and importance of corrected transposition is due to the advent of cardiac catheterization and angiocardiographic techniques for diagnosis and to the surgical correction of associated defects made possible with the advent of open-heart surgery.

Because the course of circulation is not altered in the pure form of corrected transposition the abnormality may be associated with no symptoms. However, cardiac anomalies which disturb the hemodynamics often accompany this defect. The associated anomalies thus acquire special importance in any consideration of corrected transposition. The most frequently observed anomalies are ventricular septal defect,[1,4] pulmonary stenosis,[1,5] insufficiency of the left atrioventricular valve,[4,6,7] and atrioventricular block.[1,4]

We are presenting 5 new cases seen consecutively at the Children's Hospital of the District of Columbia between 1964 and 1967 that illustrate the wide clinical spectrum with which corrected transposition of the great vessels may be associated. The clinical findings are summarized in table 1; catheterization data are listed in table 2; and electrocardiographic findings are given in table 3.

TABLE 1: Summary of Findings in 5 Patients with Corrected Transposition of the Great Vessels

Case	Age	Sex	Confirmation	Associated Anomalies		
				VSD	MI	Other Cardiac Features
1. G. H.	4 yrs.	M	Catheterization	0	0	PS, mild
2. C. B.	1 wk.	F	Catheterization	+	0	PS, L-to-R shunt
3. D. W.	5 mos.	M	Catheterization	+	0	Pulmonary hypertension
4. D. K.	3 wks.	M	Catheterization, postmortem	+	+	ASD, L-to-R shunt, PDA, LSVC
5. T. J.	6 yrs.	M	Catheterization	0	0	Complete heart-block

VSD: Ventricular septal defect
MI: Mitrial insufficiency
PS: Pulmonary stenosis
ASD: Atrial septal defect
PDA: Patent ductus arteriosus
LSVC: Left superior vena cava

Report of Cases

Case 1. G. H., a 4-year-old white boy, was admitted for evaluation of a cardiac murmur which had been noted 4 months earlier. He had no cardiovascular symptoms. Physical examination revealed a well-nourished boy in no distress. Peripheral pulses were normal, and there was no cyanosis. The heart did not appear to be enlarged. Auscultation revealed a markedly accentuated single second sound along the left sternal border with a grade 2 systolic ejection murmur best heard at the second and third left intercostal spaces. X-ray examination of the chest showed the heart to be of normal size and configuration. An electrocardiogram revealed a normal mean electrical axis. A deep QS

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* Department of Cardiovascular Surgery, Georgetown University Hospital, Washington, D. C. 20007.

† Associate in Cardiology, Children's Hospital of the District of Columbia; Assistant Professor of Pediatrics, George Washington University School of Medicine.

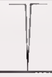
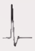
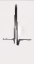
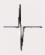
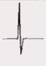
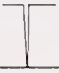
‡ Chief of Cardiology Section, Children's Hospital of the District of Columbia, 2125 13th Street, N.W., Washington, D. C. 20009; Associate Professor of Pediatrics, George Washington University School of Medicine.

TABLE 2: Catherization Data in 5 Patients with Corrected Transposition of the Great Vessels

Case	Age	Pressures (mm. Hg)					Shunt		Resistance (mm. Hg/L/m²)		Cardiac Index (L/min./m²)	
		RA	RV	PA	PC	Aorta	Direction	Site	Systemic	Pulmonary	Systemic	Pulmonary
1. G. H.	4 yrs.	m = 3	26/4	16/10	a = 6 v = 8 m = 4	105/75	0	0	21	2	6	6
2. C. B.	1 wk.	m = 1	78/9	NE	a = 14 v = 10 m = 10	85/58	R → L	Ventricle	9.1	—	7.1	3.7
3. D. W.	5 mos.	m = 0	60/10	60/35	m = 16	102/60	L → R	Ventricle	18	2.6	4	20
4. D. K.	3 wks.	m = 7	70/10	75/20	a = 9 v = 21	85/45	L → R	Atrium and ventricle	14	3	3.7	10.0
5. T. J.	6 yrs.	m = 2	35/8	30/10	—	110/50	0	0	16.5	2.3	6.2	6.2

m: Mean
RA: Right atrium
RV: Right ventricle
PA: Pulmonary artery
PC: Pulmonary capillary wedge

TABLE 3: Electrocardiograpic Features in 5 Patients

CASE	AGE	QRS AXIS	A-V BLOCK	QRS PATTERN V₁	V₆
1	4 yrs.	30°	—		
2	1 wk.	60°	—		
3	6 mos.	100°	—		
4	3 wks.	130°	1st° 2nd° 3rd°		
5	6 yrs.	-30°	3rd°		

pattern in the right-chest leads suggested a diagnosis of anteroseptal myocardial infarction. No Q wave was demonstrated in the left-chest leads.

At catheterization the only abnormality was found to be mild pulmonary stenosis with a gradient of 10 to 15 mm. Hg. Angiocardiogram (figure 1-A) demonstrated the aorta to arise ventrally and to the left from a ventricle which had the anatomic characteristics of the right ventricle. The pulmonary artery (figure 1-B) was seen to arise to the right and dorsally from a ventricle with the anatomic features of the left ventricle. The boy has remained asymptomatic and has continued to do well.

Case 2. C. B., a 1-week-old white female, was admitted because of cyanosis since birth. She was cyanotic and had a heart rate of 160 per minute. Her heart was not enlarged. A prominent single second heart

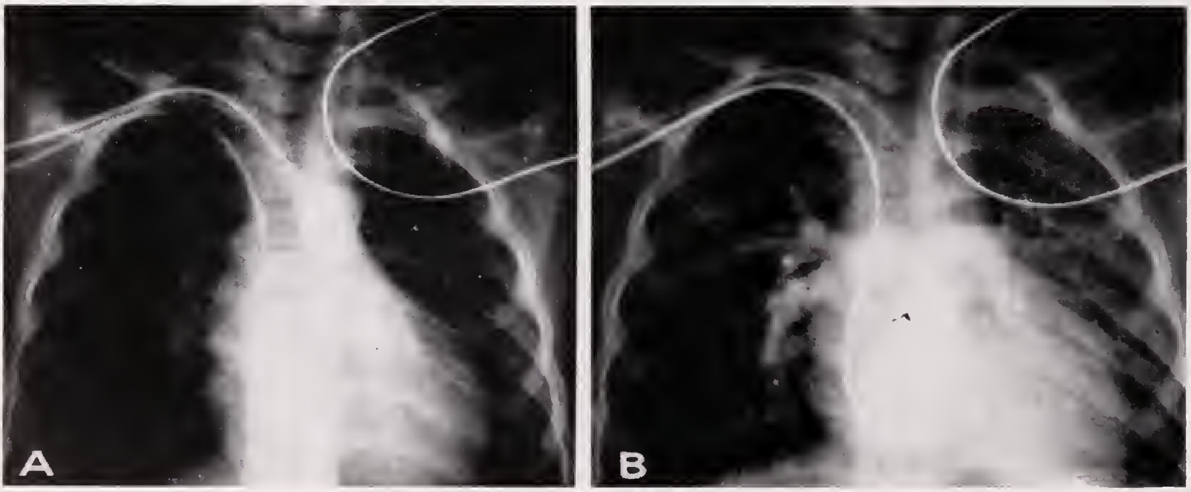


FIG. 1. A. Aortogram in G. H. showing aorta arising from area usually occupied by pulmonary artery. B. Right ventriculography demonstrating pulmonary artery arising to the right from area of normal aorta.

sound was audible along the left sternal border. A grade 4 stenotic systolic murmur was heard at the lower left sternal border. Chest X-ray films (figure 2) showed diminished pulmonary vascularity with a normal-sized heart. The electrocardiogram revealed QR in V 1 diagnostic of right ventricular hypertrophy. Cardiac catheterization and angiocardiogram established the diagnosis of corrected transposition of the great vessels with pulmonary stenosis, ventricular septal defect, and right-to-left shunt. In view of her symptoms operative treatment was undertaken and the left subclavian artery was anastomosed to the left pulmonary artery (Blalock-Taussig). Cyanosis improved. She has gained weight and has remained well since the operation.

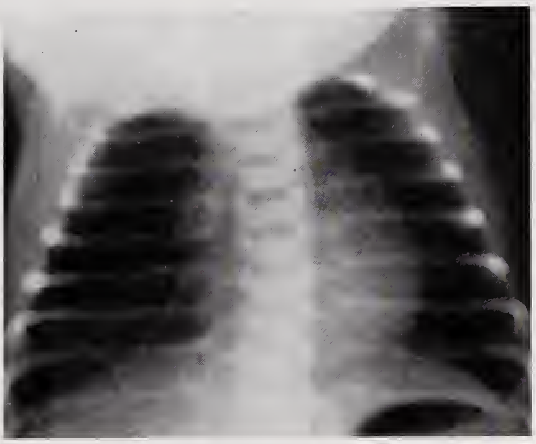


FIG. 2. Posterior-anterior X-ray film of C. B. showing normal heart size with a long, sweeping convexity to left heart border typical of corrected transposition. Note diminished pulmonary vascularity.

Case 3. D. W., a 6-month-old white male, was admitted for evaluation of slow weight gain and a cardiac murmur which had been present since birth. He had no cyanosis. His heart was hyperactive with a prominent right ventricular impulse. The second sound was single and accentuated over the upper left sternal border. A grade 3 systolic murmur and an early and mid-diastolic murmur were audible along the lower left sternal border with a fourth sound at the left sternal border. The liver was palpable 4 cm. below the right costal margin. An electrocardiogram indicated right ventricular hypertrophy as manifested by a QRS in V 1 with no septal Q in V 6. X-ray films of the chest (figure 3) showed pulmonary vascular engorgement with a supracardiac shadow suggestive of total anomalous pulmonary venous return. Catheterization findings were consistent with the diagnosis of a ventricular septal defect with a large left-to-right shunt, pulmonary hypertension, and elevated pulmonary capillary pressure. He subsequently developed recurrent atelectasis and infiltration in the right middle lobe. A bronchogram revealed obstruction of the right middle lobe which was removed with temporary improvement. He was soon readmitted for respiratory infection. Increasing right ventricular hypertrophy on the electrocardiogram led to the decision to repeat cardiac catheterization. This procedure confirmed the presence of a ventricular septal defect, large left-to-right shunt, and pulmonary hypertension. Angiocardiograms revealed the aorta arising anteriorly and to the left, with the pulmonary artery arising posteriorly and to the right, characteristic of corrected transposition. In view of the symptoms and high pulmonary artery pressure and flow, banding of the pulmonary artery was carried out with the reduction of pulmonary artery pressure to 32 to 45 mm. Hg. The baby improved markedly following the operation. Total correction is to be undertaken at 5 to 6 years of age.

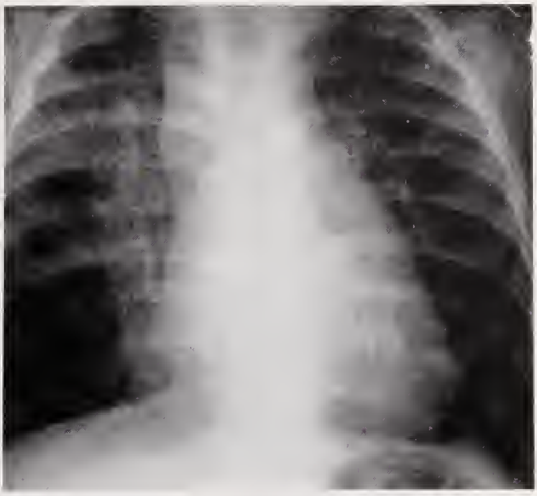


FIG. 3. X-ray film of D. W. revealing convexity of left heart border as well as active and passive pulmonary vascular engorgement.

Case 4. D. K., a 3½-week-old white male, was noted to have a single umbilical artery at birth but was otherwise considered normal. At 6 weeks, after a period of apparently normal growth, he suddenly became cyanotic and appeared septic. Physical examination revealed weak peripheral pulses, cyanosis, and rapid respirations. His heart was enlarged with a prominent right ventricular impulse and a grade 2 short systolic ejection murmur along the lower left sternal border. The liver was palpable 4 cm. below the right costal margin. A chest X-ray film showed cardiomegaly with prominence of the right atrium and ventricle. The superior mediastinum appeared widened, possibly due to a vascular anomaly. The lungs showed active congestion. An electrocardiogram was suggestive of right ventricular hypertrophy with no septal Q recorded in the left chest. Catheterization and angiocardiogram established the diagnosis of corrected transposition of the great vessels with insufficiency of the left atrioventricular valve. The baby was also found to have atrial and ventricular septal defects with a persistent left superior vena cava. He was discharged following adequate control of congestive heart failure with digitalis and diuretics. Subsequently cyanotic spells recurred and he was hospitalized twice for congestive failure associated with varying degrees of first or second degree atrioventricular block. On his third and last admission he was moribund. Respiratory distress progressively worsened and complete heart-block supervened before death finally occurred. Autopsy confirmed the catheterization findings (figure 4, A and B). The left-sided atrioventricular valve was found to be tricuspid with the septal leaflet hypoplastic and adherent in part to the septum. This resulted in a subluxation of this leaflet into the cavity of the left ventricle rendering it insufficient. He had bilateral, double renal arteries.

Case 5. T. J., a 6-year-old white male, was admitted for complete heart-block which had been recognized 2 months before admission. He had no cyanosis or clubbing and had bounding peripheral pulses. The pulse rate was 48 per minute and occasional cannon 'A' waves could be seen in the jugular vein. His precordium was prominent with palpable right and left ventricular impulses. S 1 was variable and S 2 split at the second left intercostal space. A grade 3 systolic murmur was heard over the third and fourth left intercostal spaces and grade 1 to 2 diastolic rumble was heard at the apex. An electrocardiogram showed left axis deviation, complete heart-block, and absence of a septal Q wave in the left-chest leads with prominent QS in the right-chest leads. X-ray examination revealed 'box shape' configuration of the heart (figure 5). Catheterization failed to show any evidence of intracardiac shunts, and the pressures were found to be normal. The characteristic appearance of the ventricles and the relative positions of the great vessels on the angiocardiogram confirmed the diagnosis of corrected transposition of the great vessels. The boy has remained asymptomatic to the present time.

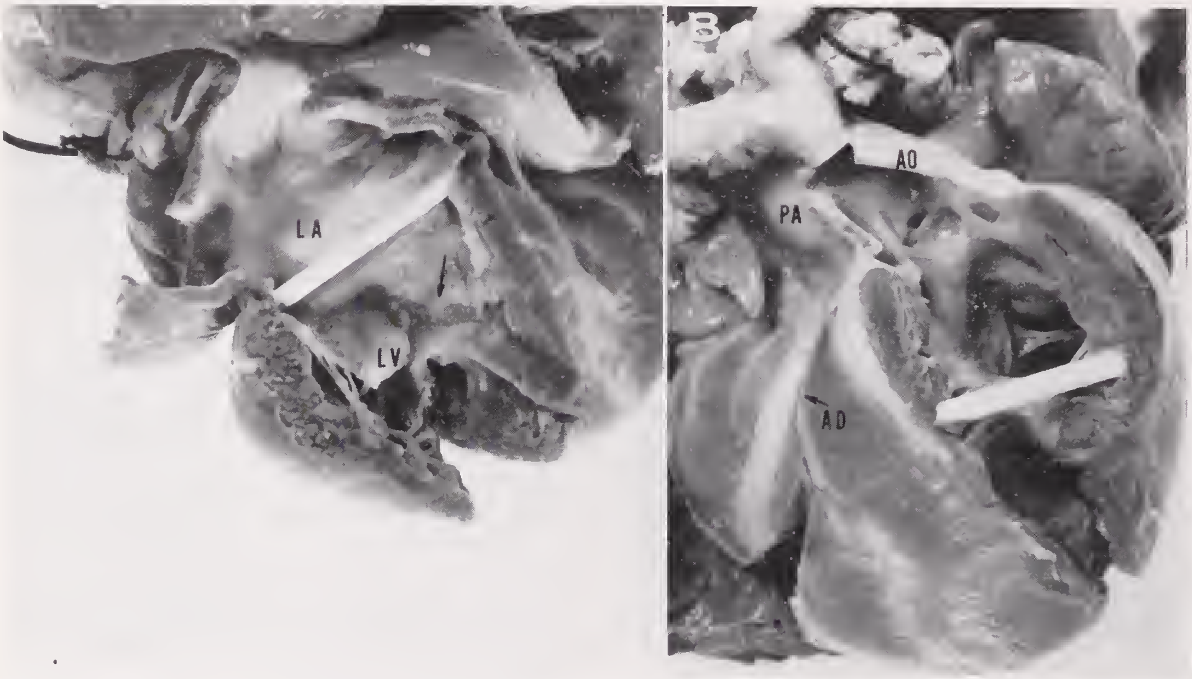


FIG. 4. A. Left-sided atrioventricular valve ring of D. K. held open by strut. Arrow points to adherence of proximal portion of septal leaflet of tricuspid valve to septal wall. The free margin of this leaflet was thus downwardly displaced into the deeply trabeculated left ventricle rendering the valve insufficient. (LA: left atrium; LV: left ventricle)

B. Left ventricle, deeply trabeculated, giving rise to aorta which arises anterior and to left. Note pulmonary artery arising posterior and to left of aorta.

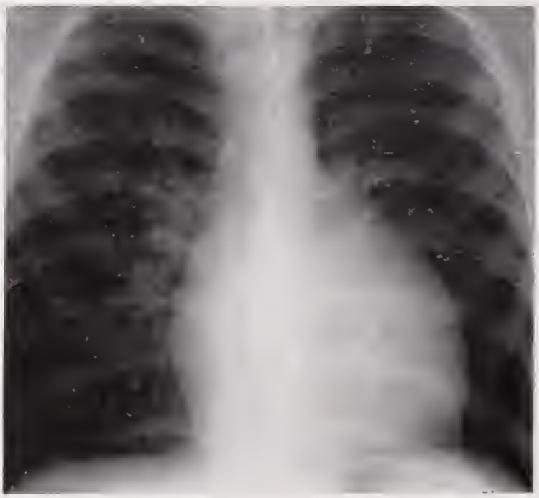


FIG. 5. X-ray film of T. J. showing considerable cardiomegaly believed secondary to complete heart-block. Convexity of left heart border with inability to define main pulmonary artery segment is characteristic of corrected transposition.

Discussion

General. The basic features of the common type of corrected transposition of the great vessels are the normal position and venous connections of the atria and the abnormal origins of the aorta and pulmonary artery from inverted ventricles which have the anatomic characteristics of the right and left ventricles respectively (figure 6, A and B).

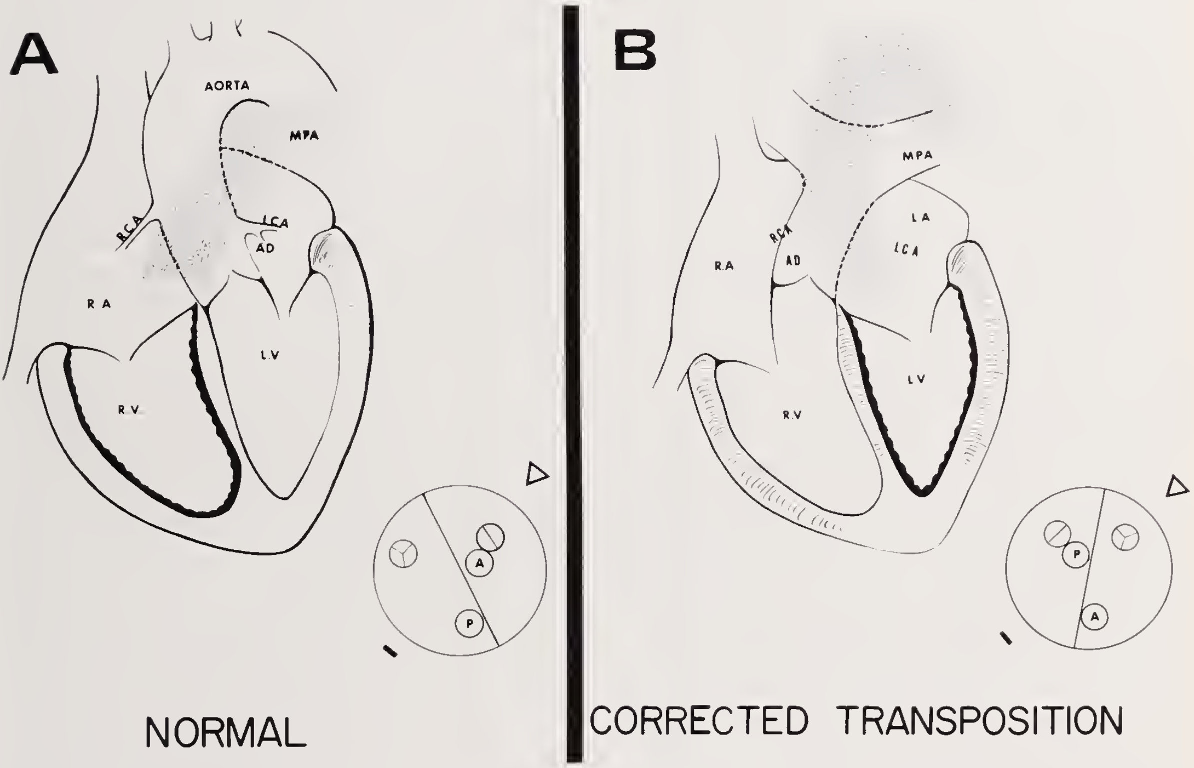


FIG. 6. A. Schema indicating normal relationship of great vessels as they relate to the ventricles from which they arise. Shown also is relationship of atrioventricular valves to their respective ventricles. The deeply trabeculated right ventricle is indicated by corrugated inner lining. Lower right hand insert is view of transected chest, viewed from above. Shaded great vessels indicate their anterior position. (MPA: main pulmonary artery; RCA: right coronary artery; LCA: left coronary artery; AD: anterior descending artery; RA: right atrium; RV: right ventricle; LV: left ventricle. Insert—A: aorta; P: pulmonary artery; ▬: sternum; ▽: vertebral body)

B. Schema of heart in corrected transposition. Note aorta arising anterior and to left with pulmonary artery arising posterior and to right, deeply trabeculated systemic ventricle, and atrioventricular valve inversion.

In view of its widespread usage, not withstanding its erroneous implication that the transposition is fully corrected, the term "corrected transposition" is used in this report. The anomaly may be superimposed on a normal situs solitus heart (heart and abdominal viscera normally placed) or in a situs inversus heart, resulting in 2 different types of corrected transposition as pointed out by Schiebler et al.[4] All of our patients belong to the first category, which is by far the commoner variety. In this type the right atrium empties into the right-sided ventricle through a bicuspid right atrioventricular valve which is essentially a mirror image of the normal mitral valve. The interior of the right-sided ventricle is conspicuous by the absence of the crista supraventricularis and the presence of fine trabeculations, i.e., the usual anatomic characteristics of the left ventricle. This chamber is also located more dorsally than in the normal heart. The left atrium receives the pulmonary veins and communicates with the left-sided ventricle through a tricuspid valve which is a mirror image of the normal tricuspid valve. The left-sided ventricle anatomically resembles the right ventricle of the normal heart in having coarse trabeculations, a crista supraventricularis, and an infundibulum. The aortic valve occupies a higher and more ventral position which corresponds to the position of the normal pulmonary valve. Although the coronary arteries arise from the aorta, they are inverted, which accounts for the course of the left coronary artery across the root of the pulmonary artery and anterior aspect of the right-sided ventricle. The normal course of circulation is therefore undisturbed in uncomplicated corrected transposition of the great vessels because the atrium receiving systemic venous blood delivers it to the pulmonary artery and the atrium receiving pulmonary venous blood delivers it to the aorta.

Embryologic considerations. There is still no general agreement regarding the embryologic basis of corrected transposition of the great vessels, although the subject has been debated for many years.[2, 8, 9] The theory of de la Cruz et al[3] has found wide acceptance and provides a clue to the understanding of the anomalies. According to these authors, the anatomic location of the right ventricle in the left-sided position and of the left ventricle in the right-sided position is a result of the development of the bulboventricular loop whose convexity is directed to the left rather than to the right. Since the atrial portion of the heart is "anchored," the position of the loop does not affect the location of the atria. Thus it comes about that the right atrium communicates with the left ventricle and the left atrium with the right ventricle. The emergence of the aorta from the anatomic right ventricle and the pulmonary artery from the anatomic left ventricle, according to this theory, is a consequence of the failure of the truncoconal septum to undergo the normal spiral rotation. The failure of rotation is also responsible for the straight character of the septum which allows the ventrally placed aortic valve to communicate with the anatomic right ventricle and the dorsally placed pulmonary valve to communicate with the anatomic left ventricle.

Prenatal and family history. The prenatal and family histories are not usually helpful in making a diagnosis. In the present series the prenatal history was normal in all patients except case 2 (C. B.) whose mother developed hypertension and infection during pregnancy. All patients were born at full term and had normal birth weights. Except for case 3 (D. W.) all babies were born in the last 4 months of the year, a finding which is in agreement with that reported by other workers.[1, 4] Neither the parents nor the siblings of any of our patients were known to have congenital heart disease. Four of the 5 patients were males. This high incidence in the male has been observed in other larger series.[1, 4] Cardell,[2] however, found the incidence in the literature to be equal among both sexes.

Clinical features. In the absence of associated anomalies, corrected transposition of the great vessels may be completely asymptomatic. Schiebler et al[4] reported the case of a man who had no symptoms and died at the age of 60 years of unrelated causes. Schaeffer and Rudolph[10] reported the case of a patient who remained asymptomatic until the age of 36 years when heart-block and progressive congestive heart-failure supervened. In the present series, case 1 (G. H.) was free from symptoms, and the cardiac murmur which led to the investigations was noted on a routine examination. Cyanosis, failure to gain weight, repeated pulmonary infections, and congestive heart failure are the more common manifestations, as illustrated by cases 2, 3 and 4 (C. B., D. W., and D. K.) in our series. These problems, however, were caused by associated anomalies, namely, severe pulmonary stenosis with a large ventricular septal defect and a ventricular septal defect with an insufficient left atrioventricular valve. The association of complete heart-block with corrected transposition is well recognized, and case 5 (T. J.) in the present series is an example. He was found to have complete heart-block, although he was completely without symptoms. Heart-block may be surprisingly well tolerated by infants. Walker et al[11] successfully repaired the ventricular septal defect in 3 patients with heart-block associated with corrected transposition of the great vessels.

It is seldom possible to recognize corrected transposition on the basis of physical examination alone. A prominent single second heart sound at the second left intercostal space may be commonly heard and is produced by the closure of the aortic valve which is located higher and more anteriorly in the position normally occupied by the pulmonary valve. This sign is, however, not constant and was noted in only 3 of our patients (cases 1, 2 and 3). The presence of complete heart-block with a history of congenital heart disease is highly suggestive of corrected transposition (case 5). Often the physical findings of the associated defects such as ventricular septal defect, insufficiency of the left atrioventricular valve, and pulmonary stenosis may be the only recognizable signs. A high index of suspicion is necessary if corrected transposition is not to be missed, especially when associated lesions overshadow the underlying anomaly. As observed by Schiebler et al[4] associated noncardiac defects are rare, and we observed only 1 such instance in this series, that of a single umbilical artery associated with bilateral double renal arteries in case 4.

Radiologic features. The more medial location of the origin of the left pulmonary artery may result in the absence of the pulmonary artery segment along the left cardiac border. The centrally placed and posterior pulmonary artery may also cause an impression on the left side of the esophagus in a barium swallow. A typical finding is an unusually straight or bulging left heart border due to the abnormal position of the root of the aorta.[12] If the left atrium is enlarged due to the insufficiency of the left atrioventricular valve or increased left atrial flow, the roentgenologic signs of left atrial enlargement may be evident. The radiologic signs are greatly altered by the presence of associated anomalies. Pulmonary vascularity, for example, may be greatly increased with a large ventricular septal defect (case 4, D. K.) or decreased with severe pulmonary stenosis (case 2, C. B). The extent of cardiomegaly is also dependent on the associated defects and the degree of heart failure. The radiologic features were described in detail by other investigators.[1, 4, 12] In our experience X-ray examination of the chest proved to be of limited value in diagnosing corrected transposition although it did suggest the presence of associated defects.

Electrocardiographic data. The electrocardiogram in corrected transposition of the great vessels may be influenced by specific-chamber hypertrophy due to associated defects. The electrocardiographic abnormalities are frequently associated with varying degrees of atrioventricular block and with reversed direction of the initial forces of ventricular depolarization.

Complete atrioventricular block (case 5, T. J.) may be due to the complete separation of the atrioventricular node and the bundle of His or to fibrosis in the main stem of the atrioventricular node. Incomplete atrioventricular block, i. e., first and second degree block, may be due to a less extensive pathologic process. The longer pathway traversed by the main bundle from the atrioventricular node to the base of the ventricular septum may predispose it to structural changes.[11, 13] Progressive degrees of heart-block are often seen and may be due to progressive fibrosis of the atrioventricular node. Other abnormalities in rhythm may include atrial fibrillation and paroxysmal tachycardia.

One of our patients (case 1, G. H.) had first degree heart-block and another (case 5, T. J.) had complete atrioventricular block. Case 4 patient (D. K.) developed varying degrees of heart-block prior to death.

The QRS axis may be normal, to the right, or to the left.[14] In 4 of our patients the axis was normal and in one (T. J.) left axis deviation was present. The reversed direction of initial ventricular depolarization forces is thought to be due to the mirror image arrangement of the conduction system which accompanies the inversion of the ventricles in this abnormality. The atrioventricular node and bundle of His have a mirror image position and the anatomy of the right and left bundle-branches is inverted.[14] This was the only constant electrocardiographic abnormality in all of our patients. All had absence of a Q wave in lead 1 and the left chest leads associated with a QR or QS pattern in V 4R or V 1.

Cardiac catheterization and angiocardiography. Cardiac catheterization data from all of our patients are given in table 2. Atrioventricular dissociation which is commonly seen during catheterization of patients with corrected transposition of the great vessels was observed in one of our patients (case 4, D. K.). He developed several episodes of first, second, and third degree heart-block and atrial flutter during the procedure. Another common practical problem is the difficulty in manipulating the catheter into the pulmonary artery from the right ventricle in view of the posterior location of the pulmonary artery and the consequent need for the catheter to make a sharp turn. This difficulty has been commented upon by others[1, 15] and was encountered in one of our patients (case 2, C. B.) who was also found to have pulmonary stenosis. The relative positions of the aortic and pulmonary catheters which appear parallel in the anteroposterior view are highly suggestive of corrected transposition of the great vessels. The catheter data will also establish the presence of associated intracardiac shunts or obstructions.

The confirmatory evidence is the characteristic appearance on angiocardiograms as first described by Anderson et al.[1] A caudally placed, smooth-walled, right-sided ventricle giving rise to the pulmonary artery which is dorsal, inferior, and medial to its usual position is characteristic. Similarly, a heavily trabeculated left-sided ventricle from which the aorta arises at a superior, ventral, and lateral position compared to the normal is also pathognomonic. These findings were uniformly observed in this series and form the ultimate basis for the clinical diagnosis of corrected transposition. The diagnostic value of angiocardiography is confirmed in numerous other reports.[4, 12, 15]

Surgical considerations. Surgical treatment is applicable to corrected transposition of the great vessels only in so far as it relates to the correction of associated anomalies. The advent of open heart surgery brought the correction of many defects within the realm of feasibility, but relatively few papers have dealt with the special surgical problems.[1, 4] Anderson et al[1] pointed out the importance of recognizing corrected transposition because of its bearing on the surgical technique to be adopted in the correction of associated defects. Transventricular repair of a ventricular septal defect, the commonest associated anomaly, may pose difficult problems because of the unusual course of the 'left' coronary artery and the relatively posterior position of the right ventricle. On the other hand, right ventriculotomy may lead to damage of the anterior papillary muscle of the right atrioventricular valve.[16] Anderson et al therefore suggested a right atrial approach for the closure of a ventricular septal defect which is more readily accessible because of the absence of the crista supraventricularis in the right-sided ventricle. Patients with corrected transposition and ventricular septal defect generally have severe pulmonary hypertension, and it may be more prudent in some cases to limit the operation to the banding of the pulmonary artery. This method was employed in case 3 (D. W.).

In the correction of pulmonary stenosis, the anomalous pattern of the 'left' coronary artery across the root of the pulmonary artery and the posterior position of the pulmonary valve may bar adequate relief of stenosis although direct surgical attack is not always impossible. The difficulties inherent in the direct approach as well as the size of the infant were important factors in the decision to perform a Blalock-Taussig operation in our second case.

The presence of heart-block does not seem to add significantly to the operative risk. Walker et al[11] were able to report successful operative repair in 3 cases of ventricular septal defect with associated complete heart-block. El Sayed et al[16] reported 7 patients with corrected transposition who had undergone surgical treatment for associated defects. Four patients died. Of 3 survivors, 1 had heart-block. These authors also stressed the surgical problems posed by the inversion of the ventricles and the anomalous course of the coronary vessels. Other reports similarly attest to the high morbidity associated with major corrective attempts.[1, 12, 17] At the present time palliative procedures such as pulmonary artery banding and Blalock-Taussig anastomosis have an important role in the surgical management of patients with corrected transposition of the great vessels.

Summary

Corrected transposition of the great vessels occurs when the convexity of the bulboventricular loop is directed to the left and the normal spiral rotation of the truncoconal septum fails to take place. As a result the venous connections of the atria are normally placed, whereas the atrioventricular valves and ventricles are inverted and are a mirror image of the normal ventricles. The aorta arises anteriorly to the left from an anatomic right ventricle receiving pulmonary venous blood, whereas the pulmonary artery arises posteriorly to the right from an anatomic left ventricle receiving systemic venous blood. The normal course of the circulation is undisturbed.

Corrected transposition is most frequently associated with ventricular septal defect, pulmonary stenosis, insufficiency of the left atrioventricular valve, and varying degrees of atrioventricular block.

Five new cases of corrected transposition have been presented, 3 of which had ventricular septal defect, 1 with mild pulmonary stenosis, and the fifth with isolated complete heart-block.

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Inappropriate Lactation

Report of Three Cases

W. TABB MOORE, M.D.*

NONPUERPERAL GALACTORRHEA and amenorrhea have been described under several eponyms—Chiari-Frommel, del Castillo, and Forbes-Albright.[1,2,3] Recent reports have emphasized both the clinical spectrum that these syndromes encompass and the association of other endocrine disorders.[4,5] The purpose of this paper is to report 3 new cases, to discuss their pathophysiology in the light of recent experimental findings, and to illustrate the necessity for repeated breast manipulation in order to detect galactorrhea.

Report of Cases

Case 1. A 36-year-old white woman was seen because of amenorrhea. She had had no periods following her first pregnancy 2½ years prior to this. She had nursed her first child for 5½ months and had noted galactorrhea for several months afterwards. For the past year and a half, however, she had not noted galactorrhea. She had been seen by several physicians and had had some tests done including a BMR, a glucose tolerance test, and a 24-hour urine steroid study. She had been given a trial of Enovid® with return of menstrual flow, but when this was discontinued menses ceased. There was no history of visual field defects, severe headaches, acne, or hirsutism. On physical examination a small amount of milky fluid was expressed from both breasts. The uterus was quite small. Visual fields were normal. A lateral skull X-ray film revealed no sella enlargement. Endometrial biopsy showed a proliferative endometrium. Vaginal smear revealed 94 per cent intermediate, 4 per cent superficial, and 2 per cent basal cells. The protein-bound iodine was 3.8 and a radioactive iodine uptake was 15 per cent. Insulin tolerance test and glucose tolerance test were normal. There was a normal growth hormone response and suppression with glucose. The hydroxycorticoids were 4.0 and 7.0 mg., 17-ketosteroids 10.1 and 7.9 mg. per 24 hours, the urine gonadotrophins 23 rat units per 24 hours, and pregnanediol excretions 7, 8 and 10 mg. per 24 hours. After the completion of the tests the patient was treated with 3 ampules of Perganol® daily (human menopausal gonadotrophin) for 6 days, and on days 5, 6 and 7, human chorionic gonadotrophin equivalent to 6,600 international units of A.P.L.® The patient conceived and had a successful pregnancy terminating in the birth of twin daughters. Subsequently the patient was lost to follow-up.

Case 2. A 24-year-old white woman was seen because of amenorrhea of 6 months' duration. Prior to that she had some irregular periods. There was no history of increasing hirsutism, baldness, loss of scalp hair, decrease in vision, or severe headache, but the patient did note an increase in facial acne. She was well developed and well nourished with slight acne. The thyroid was slightly enlarged and symmetrical without a bruit. Pelvic examination revealed no adnexal masses and a small uterus. Urinalysis and blood count were normal. The blood sugar was 94 mg. and PBI was normal, 5.4 µg. Skull X-ray films revealed an enlarged sella turcica volume of 1,040 cu. mm. (figure 1). 17-Ketosteroids were 28 mg./24 hours, and 17-ketogenics were 7 mg./24 hours. Follicle-stimulating hormone was 12.8 rat units. Pregnanediol was 0.06 mg./24 hours. With dexamethasone suppression on 2 mg. a day for 2 days there was suppression of the 17-ketosteroids to less than 50 per cent of the original value and complete suppression of the 17-hydroxy-

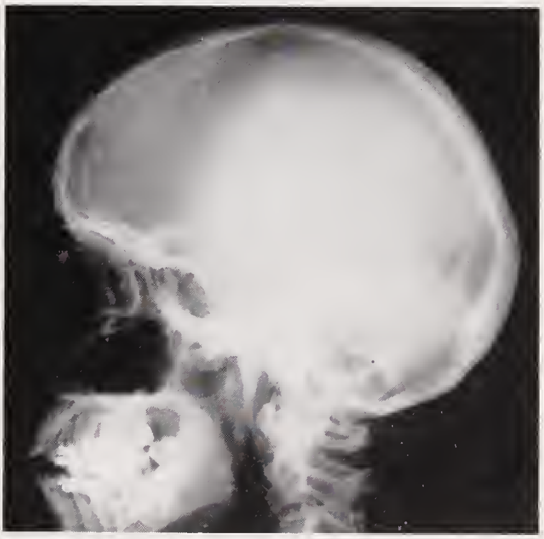

FIG. 1. Case 2. Enlarged sella turcica.

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corticoids. Suppression, however, with 8 mg. of dexamethasone revealed no further suppression of the 17-ketosteroids. Stimulation with chorionic gonadotrophin with continued dexamethasone suppression revealed no rise in 17-ketosteroids. Culdoscopy revealed normal ovaries. A pneumoencephalogram showed a slight elevation of the floor of the third ventricle, and the patient then received 3,000 R external radiation throughout bitemporal ports to the sella turcica. Subsequent to that time the patient had had no return of her periods, but on a follow-up examination milk could no longer be expressed. Follow-up endocrine studies done at the Scripps Clinic in California revealed no further changes in her thyroid or adrenal functions. The plasma cortisol was 28 and 11 μg. at 8 a.m. and 4 p.m. An 8 a.m. plasma cortisol after attempt at suppression with dexamethasone was 9.5 μg. Urine gonadotrophins at that time were 16 to 50 mouse units/24 hours. Thyroxine iodine 4.9 μg. RAI uptake was 16 per cent. A Metopirone® study at this point was normal.

Case 3. A 32-year-old woman had had no menstrual period since the delivery of a son 13 months previously. She had nursed the child for 5 months and gave no history of headaches or visual difficulties. The delivery was uncomplicated without hemorrhage or hypotension. At the time that she was seen there was a moderate amount of milky material expressible from both breasts. There were normal visual fields. Her PBI was 5.8 μg., T-3 34.4 per cent, FSH 8 and 12 rat units, 17-hydroxycorticoids 5.4 mg./24 hrs., and 17-ketosteroids 8.6 mg./24 hrs. A pregnancy test was negative. Her skull films were normal. Fifteen months postpartum the patient had a normal period and has continued to have them. There has been a disappearance of her galactorrhea.

Discussion

It has been shown by ablative and transplantation procedures that the hypothalamus secretes an inhibitory factor which prevents the secretion of prolactin from the pituitary gland.[6] When a central nervous system disturbance as seen in a pituitary tumor occurs there can be an interruption of the hypophysial portal system with a release of prolactin from the gland and subsequent galactorrhea. An inverse relationship between prolactin and FSH is probably responsible for the concomitant amenorrhea.

In the 3 cases presented in this report the chief problem concerning the patient was amenorrhea and infertility, not galactorrhea. The galactorrhea was not demonstrable except by breast manipulation, and case 2 was particularly puzzling because the first 2 attempts to demonstrate breast milk were negative. A third attempt to demonstrate galactorrhea was carried out before milk was obtained. All of the patients described illustrate that galactorrhea may not be spontaneous and that repeated attempts to elicit it must be made. The technique for expressing milk is a simple one. The breast is grasped between the hands and gentle pressure is exerted centrally. The fingers are then moved upward and inwardly toward the nipple in a milking fashion. Such manipulation should be repeated several times before one can consider that lactation is not present. This simple test in patients with amenorrhea may be more valuable than multiple steroid analysis and suppression tests. There has been little in the literature which emphasizes this important clinical point.

It has been the custom in the past to classify patients with amenorrhea and galactorrhea on a hypothalamic or pituitary basis under several eponyms. The Chiari-Frommel syndrome has come to mean postpartum amenorrhea; the del Castillo syndrome, spontaneous amenorrhea and galactorrhea; and the Forbes-Albright syndrome, amenorrhea and galactorrhea associated with a pituitary tumor, usually a chromophobe adenoma. Thus, by the previous classification 2 of our cases associated with the onset of postpartum galactorrhea and amenorrhea would be called examples of Chiari-Frommel, and the other case associated with a pituitary tumor would have been classified as Forbes-Albright. In actual fact, in only 8 of Forbes' 15 patients was a tumor demonstrated.[3] It was thought at that time that these cases were due to an increased secretion of prolactin from the pituitary gland, although Forbes considered the possibility that there was interference with an inhibitory factor from the pituitary. In view of our current knowledge the latter explanation seems to fit best with the experimental evidence.

In patients with the onset of postpartum amenorrhea and galactorrhea one can postulate that there is a small hypothalamic scar which causes a decreased secretion of the inhibitory substance. More simply, one could suppose that the delicate mechanism for resetting this regulatory control had not been made. One of our patients reset her own inhibitory regulation without therapy. In many of the cases in the literature the patients have done so spontaneously though usually after much greater lengths of time. In a recent report emphasizing the spectrum of disease involved in these syndromes the author discussed 2 patients who progressed through postpartum amenorrhea and galactorrhea (typical Chiari-Frommel) through a Forbes-Albright with pituitary tumor to the actual onset of Cushing's syndrome associated with an increased pituitary production of ACTH.[4] It would thus seem important not to classify these patients solely on the basis of an eponym but to be aware of the fact that long-term follow-up is necessary before tumor development can be ruled out and the patient can be said to have an idiopathic regulatory dysfunction. In case 2 the occurrence of a high blood cortisol, not suppressed with dexamethasone, suggests that this patient should be watched closely for the development of possible Cushing's syndrome despite her pituitary irradiation.

Several authors have discussed the association of other endocrine disorders with amenorrhea and galactorrhea. Ross et al[5] cited 4 cases of hypothyroidism, amenorrhea and galactorrhea, and raised the question of "hormonal overlap." This refers to a nonspecific stimulation of the hypothalamic pituitary system due to lack of a specific target organ hormone. VanWyk and Grumbach[7] originally described a case with hypothyroidism, precocious puberty, and galactorrhea. They postulated that because of low circulating thyroxine the patient had an overstimulation of the hypothalamus with subsequent release of TSH, a nonspecific increase in follicle-stimulating releasing factor, and interference with the release of prolactin inhibitory factor. Our case 1 had borderline low thyroid function and one would wonder if she could be an example of "hormonal overlap."

The causes of galactorrhea are listed in table 1 (modified from Dowling[8]). Those associated with drug ingestion, chest-wall irritation, and gonadal insufficiency are usually more easily diagnosed than disorders of hypothalamic or pituitary origin. Patients with a hypothalamic-pituitary disorder can often be effectively treated with Clomiphene® or human FSH to induce ovulation and reduce galactorrhea. It has therefore become increasingly important to arrive at a correct diagnosis.

TABLE 1: Causes of Galactorrhea

- I. Endocrine etiology
  - A. Physiologic postpartum lactation
  - B. Ovarian insufficiency
    - 1. Castration and partial ovarian resection
    - 2. Precocious and physiologic menopause
    - 3. Postpartum lactation and amenorrhea
  - C. Utero-ovarian tumors with or without endocrine function and surgery related to these disorders
    - 1. Carcinoma and leiomyomata of uterus
    - 2. Ovarian cystomas, carcinomas, and dermoids
    - 3. Adnexal tumors
    - 4. Hysterectomy
  - D. Other functioning endocrine tumors
    - 1. Adrenal carcinoma
    - 2. Testicular choriocarcinoma
  - E. Pituitary disease
    - 1. Acromegaly
    - 2. Chromophobe adenoma
    - 3. Hypopituitarism with galactorrhea, section of pituitary stalk
    - 4. Prolactin-secreting tumor
  - F. Hypothalamic and related disorders
    - 1. Postencephalitic parkinsonism
    - 2. Encephalitis
    - 3. Psammosarcoma of pineal body
    - 4. Pseudocyesis
- II. Local factors of etiologic significance
  - A. Mechanical stimulation
    - 1. Physiologic
    - 2. Stimulation of male or nonpuerperal female breast
  - B. Disturbances of chest wall
    - 1. Thoracic surgery
    - 2. Burns of the chest
    - 3. Herpes zoster
- III. Drugs
  - A. Hormonal substances
    - 1. Estrogen
    - 2. Androgen
    - 3. Contraceptive agents
  - B. Other drugs
    - 1. Rauwolfia alkaloids
    - 2. Chlorpromazine
    - 3. Aldomet®

One of the problems confronting the clinician evaluating patients with inappropriate lactation has been the lack of a good assay system for prolactin. The assays used for the most part have been bio-assays determining the amount of prolactin which would produce minimal stimulation of the pigeon-crop sac. Such an assay is both difficult and time-consuming. Few patients have been studied for nonpuerperal galactorrhea because of these drawbacks. Five of 7 patients evaluated by Canfield et al[9] had elevated levels of plasma prolactin. More recently attempts have been made to determine prolactin by immuno-assay. It has proven very difficult, however, to separate growth hormone and prolactin in man.[10] Glick[11] found that a preparation of human prolactin (crop-sac activity, 11.2 units/mg.) contained considerable material that cross-reacted in the immuno-assay for human growth hormone. Obviously, much remains to be learned about both the physiologic and pathologic secretion of this pituitary hormone.

Clomiphene, an analogue of the nonsteroidal estrogen chlorotrianisene (Tace®), was found to stimulate ovulation in nonovulating women. Women who have been treated with this agent have, for the most part, had to have an intact pituitary-ovarian axis. Some patients with the Chiari-Frommel syndrome have responded to its use.[12, 13]

Perganol, an extract of human FSH from postmenopausal nun's urine, was effective in inducing ovulation in our case 3. The patient subsequently gave birth to twins. Multiple births have been described with the use of this material because of the simultaneous maturation of several graafian follicles.[14] Gemzell and others have reported the successful use of these agents in the induction of ovulation in patients with amenorrhea and galactorrhea as well as other disorders.

Summary

Three cases of hypothalamic-pituitary-induced amenorrhea and galactorrhea have been presented. The importance of breast manipulation for demonstrating galactorrhea has been emphasized. The pathophysiology and the treatment of these syndromes with human FSH and Clomiphene were discussed.

The author wishes to thank Dr. Daniel D. Federman for his suggestions and help in case 2, and to Dr. Anne P. Forbes for her help in the care of case 1. The Perganol given in the latter case was administered under Dr. Forbes' direction at the Massachusetts General Hospital. I am grateful to Dr. William D. Oldham and Dr. James C. Walsh for their referral of patients.

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Radiosensitivity of Head and Neck Cancer—Sabatini

(Continued from page 643)

those in the advanced stages might benefit from radical surgery.[1, 4, 5] The above series, however, tends to demonstrate the radiosensitivity and radiocurability of many head and neck cancers. Furthermore, it demonstrates that cervical lymph-node metastases can be controlled with irradiation alone,[3] although this has not so far been our policy. These facts as well as the surgical failure often seen even in early carcinoma bring us to conclude that radiation therapy represents a very important tool in the treatment of cancer of the head and neck.

Summary

Eleven unusual cases of head and neck cancer treated with irradiation have been reviewed. The staging of the disease as well as the amount of irradiation delivered has been discussed. A follow-up is available in every case, and in all the 11 patients the disease has been successfully controlled. The value of radiation therapy in the treatment of head and neck cancer was stressed.

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Permanent Transvenous Standby (Demand) Pacemaking

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AND MICHAEL R. BOIVIN, C.P.T[‡]

THERE HAS BEEN increasing interest recently in the experimental[1] and clinical[2-6] use of the "standby" or "demand" pacemaker.[§] Indications for the clinical use of this type of pacemaker include symptomatic sinus bradycardia, symptomatic intermittent heart-block, and atrial arrhythmia with varying conduction and Stokes-Adams syndrome.

We began using an implantable transvenous endocardial standby pacemaker about a year ago for the indications listed but soon found reasons to extend our indications.[7]

Method

The "standby" or "demand" pacemaker monitors the heart rate, with the use of the same electrode for sensing and stimulating, and supplies additional beats as required to reach an acceptable rate. In one type ventricular activity inhibits the pacemaker which only stimulates if the R-R interval exceeds a preset limit.[1, 4, 6] Unfortunately, it has been difficult to shield this instrument from stray external electrical fields which may inhibit the pulse generator at a time when it should stimulate the heart, thus negating its function.[5, 8]

The second type of standby pacemaker is a modified synchronous pacemaker.[‖][5, 6] This unit is triggered by the R wave and discharges a synchronized zero delay impulse which falls harmlessly into the QRS complex. If the R-R interval exceeds a certain limit the pacemaker functions as an asynchronous unit, set at a fixed rate of 70 beats per minute. If the intrinsic R-R interval is shortened and the patient's rate exceeds 70 beats per minute, the unit follows synchronously up to 120 beats per minute, at which time an occasional R wave falls during the refractory period of the pulse generator resulting in a variable synchronous response. This becomes an exact 2:1 block in the synchronous response when the intrinsic rate attains or exceeds 140 beats per minute.

This unit cannot be inhibited by external electrical fields. It is, however, sensitive to appropriate external electrical stimuli, which may be used to study its function.[7, 9]

At first we used the permanent standby pacemaker system only in patients with symptomatic bradycardia not associated with permanent heart-block. However, one-quarter to one-third of all patients in apparent complete block revert to normal A-V conduction, at least intermittently following insertion of a pacemaker, and there is suggestive evidence that some of them die from ventricular fibrillation due to parasystole occurring during the vulnerable period.[3, 4, 6] This led us to consider the wider use of the synchronous standby pacemaker because of its greater theoretical safety factor.

We now include in the indications for a standby pacemaker all the former indications for an asynchronous pacemaker. The standby pacemaker in a patient with stable heart-block functions as a fixed-rate asynchronous unit. If A-V conduction returns, the synchronous standby pacemaker removes the danger, however small, of ventricular fibrillation resulting from competitive pacing.

The details of operative technique have been presented before and will not be discussed here, but they are important and have much to do with the subsequent success of this method.[7, 10]

Prior to the insertion of a permanent pacemaker temporary pacing is always performed as a supportive or prophylactic measure. With the insertion of the permanent unit the patient is monitored for an additional 24 to 48 hours and, barring complications, the temporary catheter is then removed and the patient allowed out of bed.

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[§] Demand Pacemaker, American Optical Company, Bedford, Mass.

Demand Pacemaker, Medtronic Inc., Minneapolis, Minn.

Standby Pacemaker ("Ventricor II"), Cordis Corp., Miami, Fla.

[‖] Standby Pacemaker ("Ventricor III" or "Ectocor") Cordis Corp., Miami, Fla.

Results

In the past year a total of 23 transvenous standby (Ectocor) pacemakers were inserted. Nineteen were inserted in as many patients as initial treatment and 4 as elective replacements for synchronous or asynchronous units in 4 additional patients (figure 1).

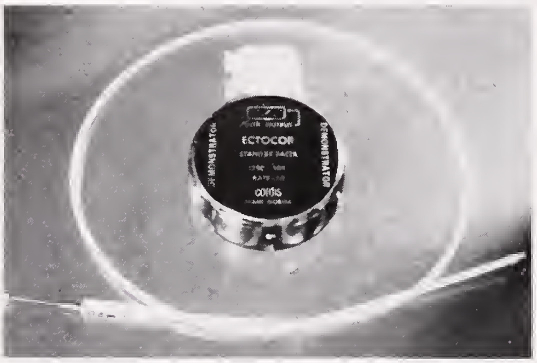

FIG. 1. Standby unipolar system. The catheter tip contains the sensing and stimulating electrode (cathode). The indifferent electrode or anode is the base plate of the pulse generator.

There have been no operative deaths, catheter dislodgments, ventricular perforations, or infections. In one patient fracture of the wire occurred close to the pulse generator 4 months postoperatively. One patient, with a highly unstable postinfarction arrhythmia with intermittent asystole and arrest, died 1 month postoperatively in ventricular fibrillation following a second myocardial infarction. The pacemaker functioned normally throughout the postoperative course. Autopsy showed the electrode to be in excellent position. The catheter was covered by a thin fibrinous sleeve throughout most of its intravascular course.

One patient 86 years old died 7 months after the replacement of a 2-year-old asynchronous unit by a standby pulse generator. Autopsy showed a recent thrombotic occlusion of the right coronary artery which apparently was the cause of death. The catheter was free of thrombus and its tip was in good position at the apex of the right ventricle.

Twenty-one patients with permanent standby pacemakers are alive and well, pacing satisfactorily.

Characteristics of the Synchronous Standby Pacer and Methods of Testing

If the patient's own heart rate exceeds that of the synchronous standby pacemaker, it is difficult to be certain that capture is present, since electrocardiographic fusion complexes will be present with or without capture (figure 2). This may be checked in one of several ways: The heart rate may be slowed by carotid arterial compression or the use of intravenous edrophonium chloride.[3] When the patient's rate drops below 70 beats per minute the altered complex of pacemaker ventricular

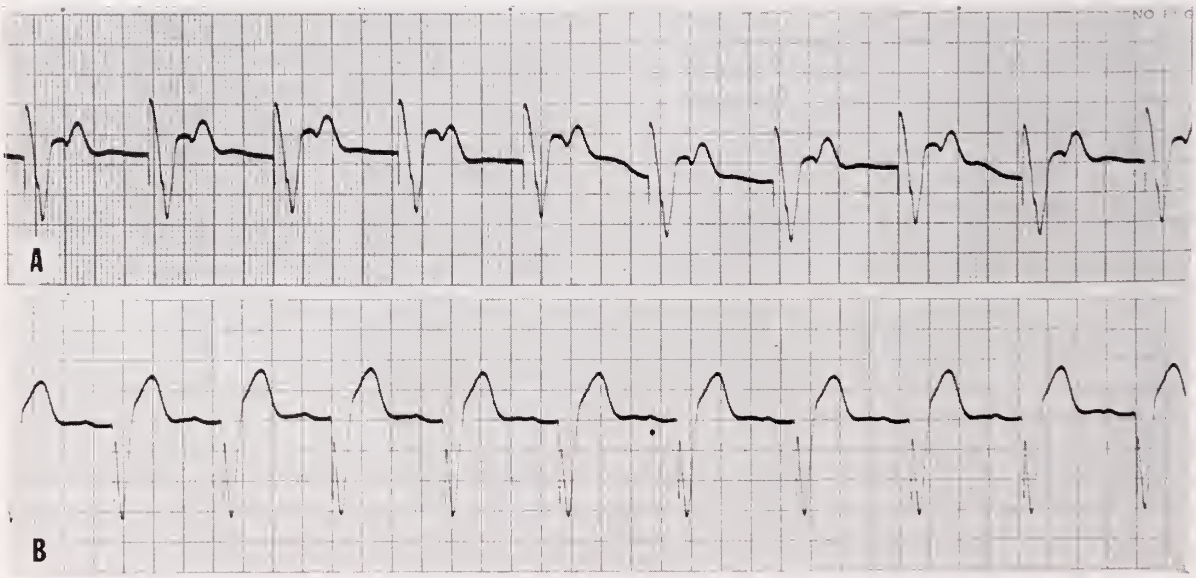

FIG. 2. (A) Lead AVF. Standby pacing. Pacer driving heart asynchronously at 72 beats per minute. (B) Lead II. Normal sinus rhythm at 80 beats per minute. Pacer following synchronously at same rate.

capture becomes apparent. This is also seen spontaneously when the patient's intrinsic rate is 70 beats per minute or close to it (figure 3).

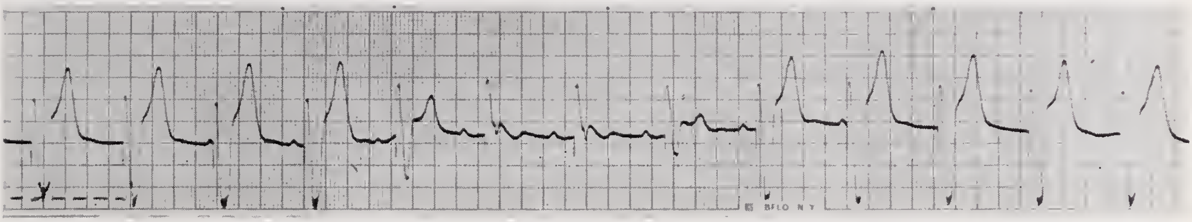

FIG. 3. Lead AVF. Standby pacing at 72 beats per minute in a patient with intermittent heart-block currently in normal sinus rhythm. The rhythm varies from the pacer rhythm at 72 beats per minute to sinus rhythm at 74 beats per minute and back again.

Alternatively, a magnetic switch in the pulse generator may be activated by external application of a magnet, blocking the amplifier in the synchronizing circuit (figure 4). The pacer then reverts to an asynchronous mode at its basic rate of 70 per minute. Competitive pacing will result, demonstrating capture if present (figure 5). Finally, the principle of "external" or "indirect overdrive" may be used.[7, 9]

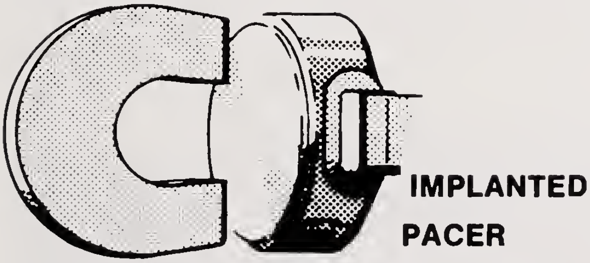

FIG. 4. Drawing showing magnet applied at right angles to magnetic switch located below the plug in the standby pulse generator.

In this technique a low amplitude impulse (5 v. and 5 to 10 ma.) from any temporary internal pacing unit is externally applied to the anterior chest at a rate faster than the patient's.[9] One electrode is placed over the pulse generator and the other over the approximate position of the ventricular electrode (figure 6). The signal is much too weak to affect the heart's rate directly, but the synchronous standby pulse generator will read it as an R wave and synchronize on this faster external stimulus. If capture is present the "actual" pulse rate will also increase appropriately (figure 7). If both techniques, magnet and overdrive, are combined, an even more dramatic change in the electrocardiogram results (figure 8). The overdrive principle may also be used to test the variable and exact 2:1 block in pacer synchronization.

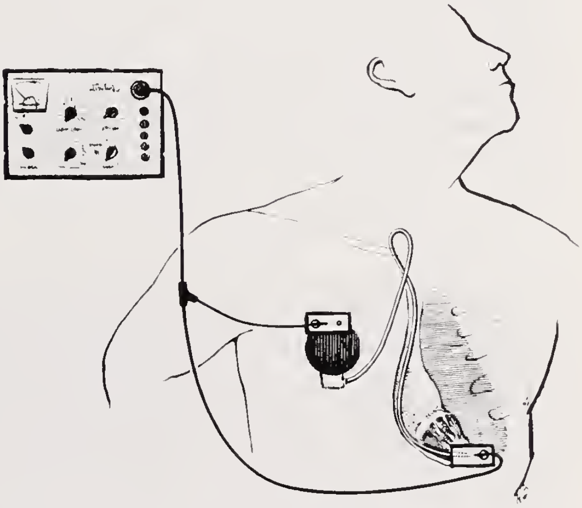

FIG. 6. Drawing of system used for external, or indirect overdrive, showing position of skin electrodes.

The mechanism of variable and 2:1 block in

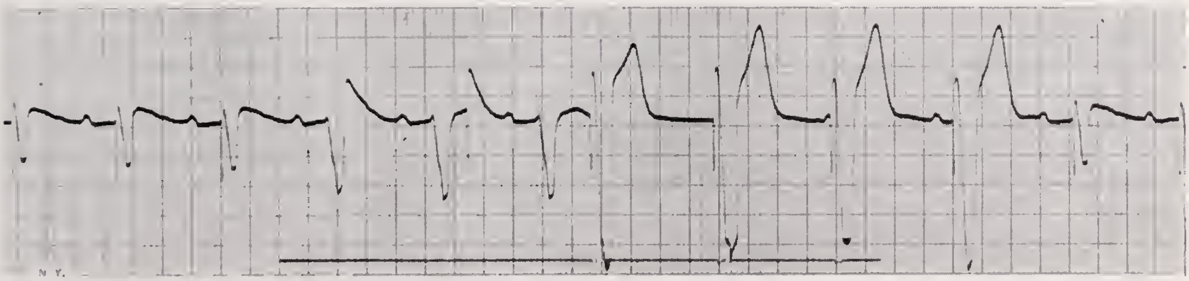

FIG. 5. Lead AVF. Heart rate 80 beats per minute. Pacer is following synchronously. Line marks magnet application. Rhythm changes to competitive pacing.

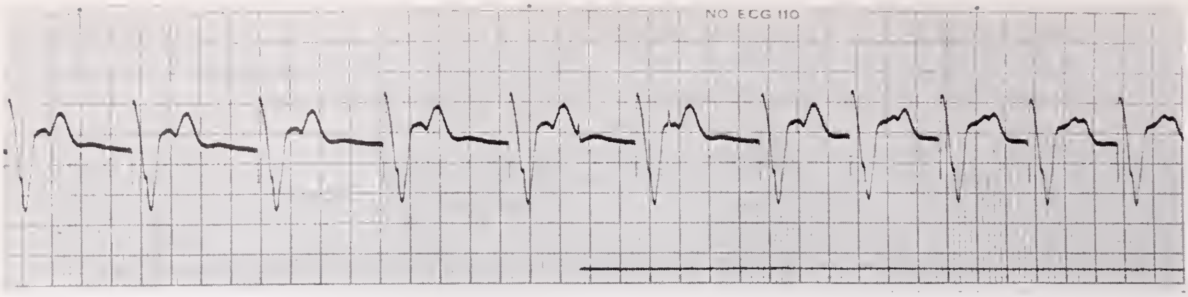

FIG. 7. Lead AVF. Standby pacing at 72 beats per minute. Line indicates application of "indirect overdrive." Pacer follows overdrive signal at 100 beats per minute.

pacer synchrony is as follows: In the asynchronous pacemaker state the pulse generator emits an impulse at a firing frequency of 0.86 seconds, and following each impulse there is a built-in delay or refractory period of 0.5 second during which time-pacemaker firing is impossible.

Should the patient's own ventricular rate reach 120 beats per minute the intrinsic R-R interval becomes 0.5 second, equal to but not less than the refractory period of the pulse generator. Complete synchrony will therefore occur, with each R wave stimulating a pacemaker impulse.

If the patient's own ventricular rate should exceed 120 beats per minute the R-R interval then becomes less than 0.5 second and the pulse generator can no longer respond to, or synchronize with every intrinsic stimulus. This is demonstrated electrocardiographically by 3 characteristic complexes in sequence: an intrinsic ventricular complex with a synchronized pacemaker spike,

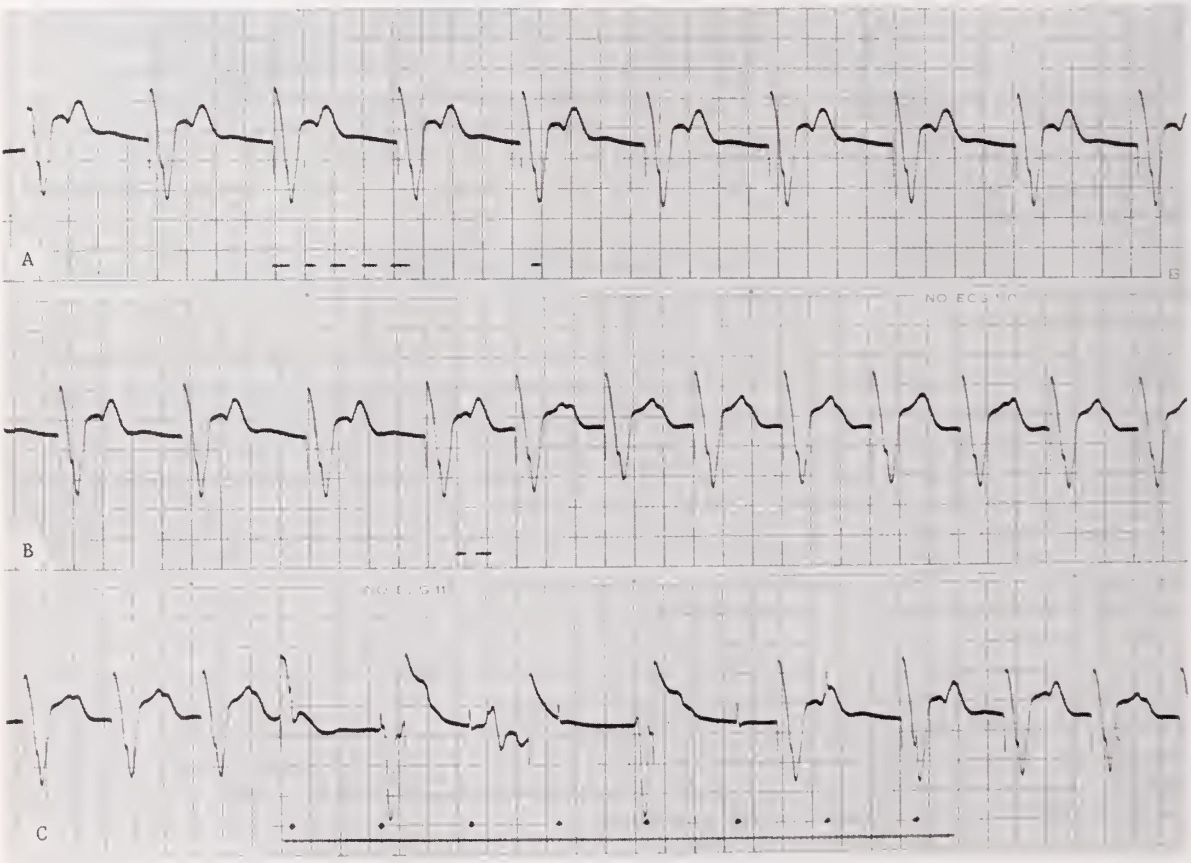

FIG. 8. Results of blocking synchronizing circuit are even more striking at "overdrive"-induced faster rate. Lead AVF. (A) Standby pacing at rate of 72 beats per minute. (B) "Indirect overdrive" rate 100 beats per minute starting at marker. (C) Magnet application indicated by marker. Dots indicate ineffective "overdrive" signals. Competitive asynchronous pacing of pacer and patient results.

followed by an intrinsic ventricular complex without a synchronized pacemaker spike, followed by a third complex which is a pacemaker-induced ventricular complex. This cycle may then be repeated as long as the patient's heart rate falls between 120 and 140 beats per minute. At or above a rate of 140 beats per minute the pulse generator can only respond to alternate stimuli resulting in a 2:1 block in the synchronous relationship (figure 9).

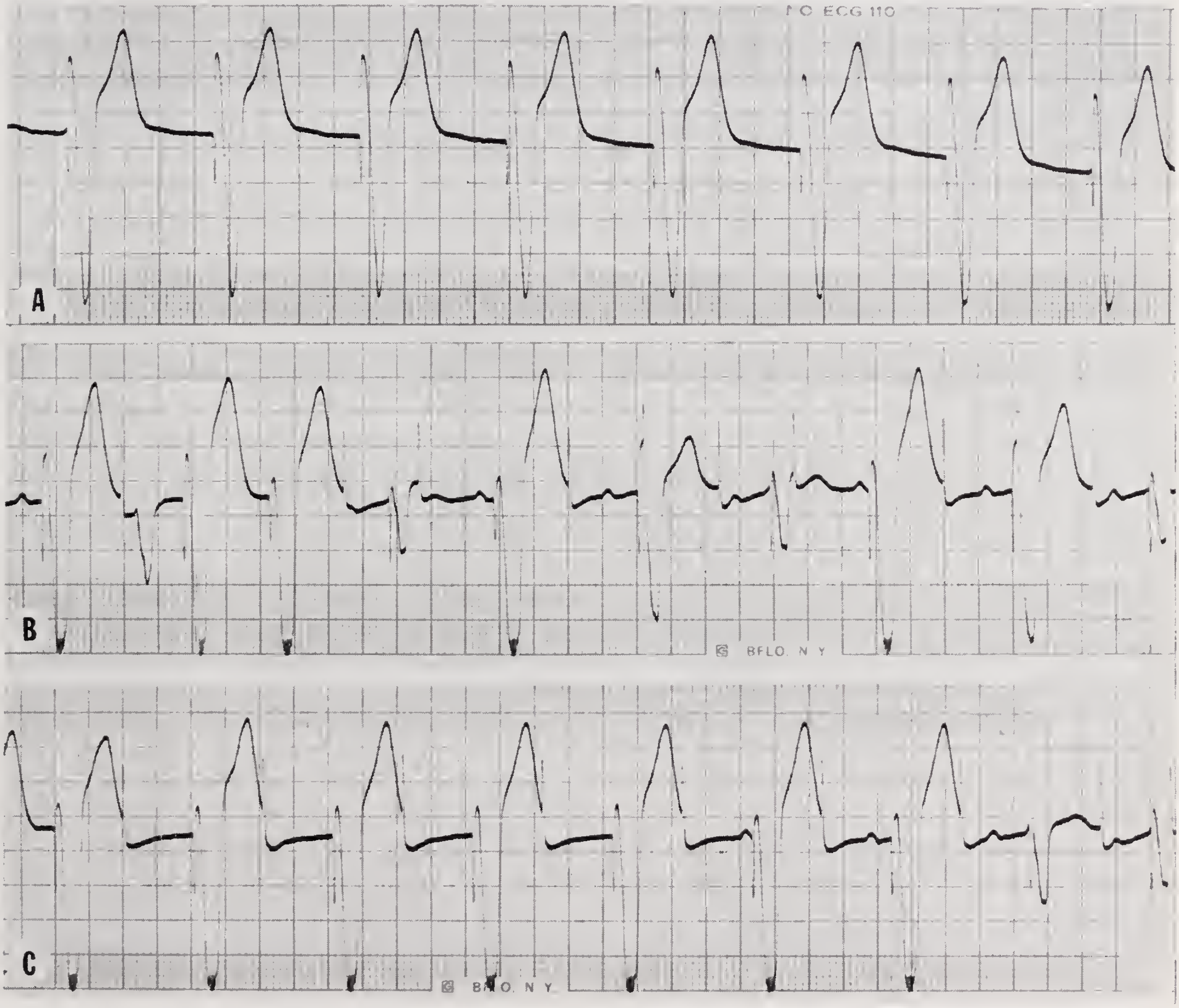

Fig. 9. Lead AVF. (A) Initial rate of 72 beats per minute. (B) Partial 2:1 block at rate of 130 beats per minute induced by indirect overdrive. (C) Complete 2:1 block at rate of 150 beats per minute induced by indirect overdrive.

Only intrinsic heart rates of 120 to 140 beats per minute with their attendant arrhythmias will allow this form of parasystole to develop. Under the artificial conditions of indirect overdrive, while improbable, it is possible for a pacemaker stimulus to fall on the ascending limb of the T wave (figure 10).

At the present time the potential danger of this situation seems less than that of the unwanted inhibition of the other type of demand pacemaker by stray external electrical fields. However, as adequate protection against outside interference is developed, the inhibitory type of demand pacemaker will be the preferred unit, since it produces no distortion of the QRS complex and should have longer battery life.[5]

Since the danger of competitive pacing is eliminated, the demand unit seems preferable to the fixed rate (asynchronous) unit in complete heart-block and is specifically indicated in all other types of symptomatic bradycardia.

The simplicity and safety of the transvenous method make it the method of choice, especially in older patients.

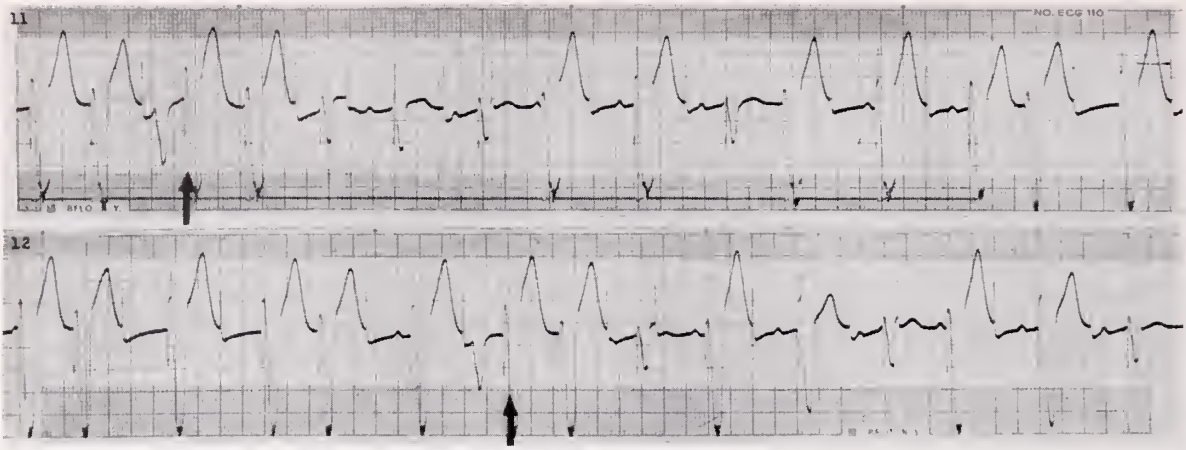

FIG. 10. Lead AVF, showing indirect overdrive stimulation at a rate of 130 beats per minute. Incomplete 2:1 block results, showing an irregular rhythm, with the pacemaker firing in the ascending limb of the T wave at points indicated by arrows.

Summary

The transvenous system of permanent pacemaking is simple and safe and is preferred for all older patients.

The standby (demand) pacemaker functions in all types of symptomatic bradycardia, virtually eliminating the danger of competitive pacing and is, therefore, preferred to the asynchronous (fixed-rate) pacemaker.

The continuously functioning synchronous standby (demand) pacemaker is currently preferred to the intermittently functioning inhibitory or circuit blocking type because of the danger of pacer inhibition by stray electrical fields in the latter.

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Indications for Cardiac Pacing in Acute Myocardial Infarction*†

PHILIP SAMET, M.D.,‡

Congestive heart failure, shock, and cardiac arrhythmias with rapid or slow ventricular rates are 3 complications of acute myocardial infarction resulting in increased mortality. The purpose of this report is to outline the role of cardiac pacing in the treatment of some of these abnormal rhythms.

Sinus bradycardia occurs in 11 to 14 per cent of patients with acute myocardial infarction. First- and second-degree atrioventricular block have been observed in 12 to 23 per cent, and third-degree or complete atrioventricular block in 2 to 8 per cent of patients with acute infarction. Complete heart-block was associated with a mortality rate of 58 to 100 per cent prior to the era of pervenous cardiac pacing. The lesser degrees of atrioventricular block are potential warnings of complete heart-block and carry a high risk of ventricular standstill, ventricular tachycardia, and ventricular fibrillation. Sinus bradycardia is probably also associated with ventricular asystole and the varied grades of atrioventricular block.

Since the initial report on the use of the pervenous temporary catheter electrode for the treatment of slow ventricular rates caused by complete heart-block complicating acute myocardial infarction, a number of observers have employed catheter pacing under similar circumstances in preference to drug therapy with Isuprel® or atropine. It must be stressed that spontaneous reversion to normal sinus rhythm occurs in the majority of patients with complete heart-block complicating acute myocardial infarction. For this reason the recent development of the standby or demand-pacing mode is of great practical importance. In this mode of pacing an effective pacing stimulus results only when a QRS complex is absent for a pre-set, predetermined interval. For example, if the demand interval is set at 60 beats/min. a pacer stimulus results if a QRS complex is absent for more than 1 second; if the demand interval is set at 30 a stimulus results if the QRS complex is absent for more than 2 seconds; if the demand interval is set at 120 per minute a stimulus results if the QRS complex is absent for more than 0.5 second. Competitive stimulation between the pacer stimuli and the patient's own QRS complex is thus avoided, reducing or abolishing the possibility of repetitive ventricular rhythms (ventricular tachycardia or fibrillation) if a pacer stimulus happens to fall on the apex of a T wave during the so-called vulnerable period. The latter may readily occur if fixed rate pacers are employed with transient complete heart-block. The demand pacer impulse is generally set at 2 to 3 times the threshold level to minimize further the possibility of repetitive ventricular firing. Demand pacers are now readily available from a number of manufacturers. External pacing plays little part today in the therapy of complete heart-block with or without the complication of acute myocardial infarction.

The technique of temporary catheter passage deserves comment. A variety of veins may be employed to afford catheter passage—the cephalic or basilic veins, the subclavian, saphenous, or external jugular veins. Since the jugular veins are often utilized for permanent catheter passage, we prefer to use the median basilic veins for temporary pervenous pacing. Catheter displacement within the right ventricular chamber is not a significant problem when arm veins are employed. We usually do not employ the blind method of passage of electrode catheters. An image amplifier fluoroscope located in the cardiac catheterization laboratory is used if the patient can be moved. If the patient cannot be moved, "blind" passage with a bipolar catheter has been attempted with the tip of the catheter as an exploring intracavity lead to check position; the latter approach is successful in 75 per cent of patients in experienced hands. A portable image amplifier is now available for bedside use and obviates the necessity for blind passage in patients who cannot be moved to the catheterization laboratory. We have utilized only bipolar catheters with the tip placed either in the right ventricular outflow tract or the right ventricular apex. The thin platinum-tipped wires recently recommended by some cardiologists are not employed because of difficulty in blind passage and the danger of knot formation. On rare occasions, in emergency situations a transthoracic wire may be utilized to permit rapid ventricular capture by direct left ventricular needle puncture and passage of the pacing wire through the needle into the left ventricle.

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This discussion refers primarily to cardiac pacing in complete heart-block. A few clinicians advocate initial therapy with atropine or Isuprel for second-degree atrioventricular block and, if these fail, cardiac pacing. We utilize initial pacer therapy as do most cardiologists. In order to avoid competitive rhythm, demand standby pacing is again the treatment of choice. Fixed rate pacers are especially contraindicated under these circumstances because of the danger of repetitive ventricular rhythms with ventricular fibrillation. On the other hand, first-degree atrioventricular block per se (without any other arrhythmia) is not treated by cardiac pacing but is continuously and carefully monitored.

Sinus bradycardia often requires pacer therapy. If the rate is between 50 and 60, atropine or Isuprel therapy is carefully utilized in our institution since these agents may slow the ventricular rate while increasing the atrial rate. If the rate falls below 50/min. a bipolar catheter is passed into the right atrium by techniques similar to those described above and set on atrial demand pacing modes at a rate of 60 to 70. Since atrioventricular conduction per se is normal under these circumstances, the ventricular rate is also 60 to 70. If any degree of atrioventricular block is present along with sinus bradycardia, the electrode tip catheter is passed into the right ventricle for right ventricular demand-pacing at a rate of 70 to 75. Nodal rhythm below rates of 60 is also treated by right ventricular demand pacing at rates of 70 to 75.

The most recent development in catheter-electrode pacing in patients with acute myocardial infarction involves the concept of overdriving. Despite the availability of digitalis, quinidine, procainamide, Xylocaine®, propranolol, etc., some patients with episodic atrial and ventricular tachycardias continue to pose a clinical problem. The cardiac-pacing catheter has been utilized both on a temporary and permanent basis to overdrive the heart (by either atrial or ventricular pacing) at rates greater than the sinus rate to prevent such ectopic rhythms. It must be emphasized that the use of the pacing catheter under these circumstances is not for patients with atrioventricular block but for patients with varied types of atrial and ventricular arrhythmias. The ultimate usefulness of this type of pacing remains to be established.

Ways to Prevent Shooting Accidents

To PREVENT accidental gunshot wounds, Mr. Robert M. Oswald, national director of American Red Cross Safety Services, says, read and remember these rules:

Never point at anything you don't intend to shoot.

Wear bright colors when hunting and know the terrain. A fall can cost you your life if you are carrying a loaded gun.

Take along a small first aid kit, a compass, knife or hatchet, and matches.

Treat every gun as if it were loaded. Never take anyone else's word that a gun is "empty."

Be sure of your target. Never shoot at noises - wait until the whole animal or bird is visible.

Know the correct way to carry your gun — under arm, shouldered, cradled, or with both hands. Make sure safety is on, finger outside trigger guard, muzzle in safe direction and under control.

Keep the safety on or the chamber unloaded until ready to fire . . . and keep your finger out of the trigger guard.

Be sure your ammunition is the right caliber and powder load for your gun.

Check bore through the breech end before loading to be sure it is clear of foreign objects. Repeat this check frequently while hunting.

Be sure the action is open when handing the gun to another person, or when stopping to eat, chat, or rest.

Transport unloaded guns in cases.

When duck hunting from a boat, 2 hunters should sit back to back. Never place a loaded gun on bottom of boat.

When hunting in a group, no hunter should carry a gun so that it ever points at another person.

Unload a gun before crossing a fence and pass gun under first.

Keep guns stored out of the reach of children and unloaded. Lock up guns and ammunition in separate places.

Teach your children the principles of firearms safety.

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Mylanta helps relieve ulcer pain with the two most widely prescribed antacids: aluminum and magnesium hydroxides.

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Mylanta *also* contains simethicone: for concomitant relief of G.I. gas distress.

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The *prolonged* acceptance of Mylanta was recently confirmed in 87.5% of 104 patients—after a total of 20,459 documented days of therapy.* *Danhof, I. E.: Report on file.

Composition: Each Mylanta chewable tablet or teaspoonful (5 ml.) contains: magnesium hydroxide, 200 mg.; aluminum hydroxide, dried gel, 200 mg.; simethicone, 20 mg. *Dosage:* One or two tablets (well chewed or allowed to dissolve in the mouth) or one or two teaspoonfuls to be taken between meals and at bedtime.

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No two women are quite alike...

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The progestin is distinctive, and for some women this may mean a different clinical response. The Compack® tablet dispenser is distinctive; its functional simplicity makes it virtually patient-proof. The acceptance of Ovulen-21 is distinctive… together with Ovulen®, it is more often prescribed than any other individual contraceptive product currently available.

Indication—Oral contraception.

Contraindications—Thrombophlebitis, thromboembolic disorders, cerebral apoplexy or a past history of these conditions, markedly impaired liver function, known or suspected carcinoma of the breast, known or suspected estrogen-dependent neoplasia, undiagnosed abnormal genital bleeding.

Warnings—Watch for the earliest manifestations of thrombotic disorders (thrombophlebitis, cerebrovascular disorders, pulmonary embolism, retinal thrombosis); if present or suspected discontinue the drug immediately.

British studies reported in April 1968[1,2] estimate there is a seven- to tenfold increase in mortality and morbidity due to thromboembolic diseases in women taking oral contraceptives. In these controlled retrospective studies, involving 36 reported deaths and 58 hospitalizations due to "idiopathic" thromboembolism, statistical evaluation indicated that the differences observed between users and non-users were highly significant. The conclusions reached in the studies are summarized in the table below:

Comparison of Mortality and Hospitalization Rates Due to Thromboembolic Disease in Users and Non-Users of Oral Contraceptives in Britain.

Category	Mortality Rates		Hospitalization Rates (Morbidity)
	Age 20-34	Age 35-44	Age 20-44
Users of Oral Contraceptives	1.5/100,000	3.9/100,000	47/100,000
Non-Users	0.2/100,000	0.5/100,000	5/100,000

No comparable studies are yet available in the United States. The British data, especially as they indicate the magnitude of the increased risk to the individual patient, cannot be applied directly to women in other countries in which the incidences of spontaneously occurring thromboembolic disease may differ.

Discontinue medication pending examination if there is sudden partial or complete loss of vision, or sudden onset of proptosis, diplopia or migraine. Withdraw medication if papilledema or retinal vascular lesions are found.

Since the safety of Ovulen in pregnancy has not been demonstrated, it is recommended that pregnancy be ruled out for any patient who has missed two consecutive periods before continuing the contraceptive regimen. If the patient has not adhered to the prescribed schedule the possibility of pregnancy should be considered at the first missed period.

A small fraction of the hormone agents in oral contraceptives has been identified in the milk of mothers receiving these drugs. The long-range effect to the nursing infant cannot be determined at this time.

Precautions—Pretreatment physical examination should include special reference to the breasts and pelvic organs, and a Papanicolaou smear.

Endocrine and possibly liver function tests may be affected by Ovulen. Therefore, it is recommended that such tests if abnormal be repeated after the drug has been withdrawn for two months.

Pre-existing uterine fibromyomas may increase in size under the influence of progestogen-estrogen preparations.

Because these agents may cause some degree of fluid retention, conditions which might be influenced by this factor, such as epilepsy, migraine, asthma, cardiac or renal dysfunction, require careful observation.

In breakthrough bleeding, and all irregular vaginal bleeding, consider nonfunctional causes. Adequate diagnostic measures are indicated in undiagnosed vaginal bleeding.

Carefully observe patients with a history of psychic depression and discontinue the drug if severe depression recurs.

Any possible influence of prolonged Ovulen therapy on pituitary, ovarian, adrenal, hepatic or uterine function awaits further study.

A decrease in glucose tolerance has occurred in a significant percentage of patients on oral contraceptives. The mechanism of this decrease is obscure. For this reason, diabetic patients should be observed carefully while receiving Ovulen.

Because of the effects of estrogens on epiphyseal closure Ovulen should be used judiciously in young patients in whom bone growth is not complete.

The age of the patient constitutes no absolute limiting factor, although Ovulen therapy may mask the onset of the climacteric.

The pathologist should be informed of Ovulen therapy when relevant specimens are submitted.

Adverse Reactions—A statistically significant association has been shown between use of oral contraceptives and the following serious adverse reactions: thrombophlebitis, pulmonary embolism.

Although available evidence is suggestive of an association, such a relationship has been neither confirmed nor refuted for the following serious adverse reactions: cerebrovascular accidents, neuro-ocular lesions, e.g., retinal thrombosis and optic neuritis.

The following adverse reactions are known to occur in patients receiving oral contraceptives: nausea, vomiting, gastrointestinal symptoms (such as abdominal cramps and bloating), breakthrough bleeding, spotting, change in menstrual flow, amenorrhea during and after treatment, edema, chloasma or melasma, breast changes (tenderness, enlargement, secretion), change in weight, changes in cervical erosion and cervical secretions, suppression of lactation when given immediately post partum, cholestatic jaundice, migraine, allergic rash, rise in blood pressure in susceptible individuals, mental depression.

Although the following adverse reactions have been reported in users of oral contraceptives, an association has been neither confirmed nor refuted: anovulation post treatment, premenstrual-like syndrome, changes in libido, changes in appetite, cystitis-like syndrome, headache, nervousness, dizziness, fatigue, backache, hirsutism, loss of scalp hair, erythema multiforme and nodosum, hemorrhagic eruption, itching.

The following laboratory results may be altered by oral contraceptives: hepatic function: increased sulfobromophthalein and other tests; coagulation tests: increase in prothrombin, Factors VII, VIII, IX and X; thyroid function: increase in PBI and butanol extractable protein bound iodine, and decrease in T^3 uptake values; metyrapone test; pregnanediol determination.

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Before prescribing see Detailed Product Information.

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If you think the pink pill for U.R.I. symptoms is built for speed and endurance you're on the right track.

Here is a tablet that begins to relieve symptoms of upper respiratory infection quickly—a tablet that works for hours to make it easy for your patient to enjoy continuous relief.

Novahistine Singlet combines effective dosage of an antipyretic-analgesic with a vasoconstrictor-antihistamine formulation to relieve not only the congestion, but also the fever and the aches and pains that almost always accompany upper respiratory infections.

A total daily dosage of 3 or 4 tablets will normally provide the continuous relief your patient expects. Use with caution in patients with severe hypertension, diabetes mellitus, hyperthyroidism or urinary retention. Caution ambulatory patients that drowsiness may result.

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Novahistine® Singlet decongestant-analgesic

(Each tablet contains: phenylephrine hydrochloride, 40 mg.; chlorpheniramine maleate, 8 mg acetaminophen, 500 mg.)

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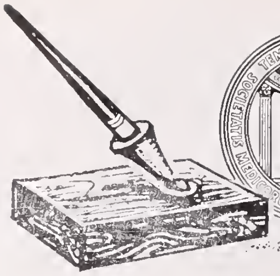

President's Page

THE REVOLUTION IN MEDICAL CARE

No practicing physician can be totally unaware of the fact that great changes are taking place in the patterns of medical care in America. But because most doctors are extremely occupied with their practices, and because the alterations which are occurring are not often visible on the surface, the extent and depth of this revolution may be greater than the profession as a whole realizes.

The causes of the changes are numerous. There has been a progressive movement of the Nation's population from the rural to urban areas. Because of this and the rapid advances in medical technology, necessitating large medical centers with trained personnel, more and more graduating medical students are turning away from general practice toward the specialties. Throughout the country there is an increasing support of medical schools and research by government funds, bringing with it tighter controls of teaching and research by Federal and state governments. Similarly, the payment of the cost of care for aged and indigent people by the Federal Government seems only the beginning of a tendency toward third-party involvement between the physician and his patients.

The results of these influences are beginning to be felt everywhere. Rural areas are finding it more difficult to find young general practitioners to replace those too old to continue their work. There is an increasing orientation of physicians and surgeons around medical centers and a greater tendency of doctors to form groups or clinics with various specialty representation. Medical school-oriented group practice has been established in Boston and New Haven, affiliated with Harvard and Yale, respectively. Similar groups are appearing in other cities. Some include provision for prepaid medical care and have been funded with the aid of government funds. The involvement of third parties between physicians and patients, whether they are the Government, insurance companies, or unions, has had the inevitable effect of fee regulations.

The obvious direction toward which all of these movements are headed is some form of controlled medicine. We cannot deny that some benefits accrue to systems of total medical coverage. Chief among these is a relief for the family from the burden of medical expenses for the care of its aged or chronically ill members. Disadvantages include a deterioration in doctor-patient relationship, less motivation of physicians, less research, government bungling, and vast government expense.

The American Medical Association, recognizing the changing patterns of medical care, has established a new Division of Public Affairs to help inform its members of the new trend. In general, our one aim must be: To Provide the Best Medical Care for All Americans.

William S McCune

Editorials

Christmas Decoration as Therapy

Responding to the need for decorating our homes and offices at Christmas and, even more important, participating in the seasonal effort, is a sign of "being with it." For example, doctors know that when patients decorate their own rooms it is a hopeful sign. It might be useful for us to remember that decorations are different things to different people. To some they are religious symbols, to others they are aesthetic sensations. But they are also a form of healing for both mind and spirit. It is a good thing that we keep up this tradition. What's good for the spirit is good for the psyche!—Adapted from *News from Sibley Hospital*, January 1968.

Z. M. L.

Genetics Counseling*

The area of medical or paramedical service known as Genetics Counseling is relatively new and, for many, still somewhat mysterious. This atmosphere exists because of the relative newness and rapidity of growth of the science of genetics and the tendency of many to exempt, in their own minds, the human organism from the blind laws of probability.

My approach to genetics counseling is nondirective. The process is aimed at decision-making, but the decision should be freely made by the client or clients in the light of the information given. As I see it, the objective of genetics counseling is to help the counselees to arrive at a reasonable decision for themselves. The decisions to be made are either marrying or not, and, if married, reproducing or not. These are such vital decisions which are fundamental to all of a person's life that someone with the limited knowledge of a genetics counselor would be extremely presumptuous to make such decisions. For one to impose his own position of authority on such decison-making is to take away basic human freedom. However, the counselor has the duty of informing the couple of the various ramifications of their decision, especially those affecting their present family and marital relationship.

Couples, pre- or postmaritally, are the usual clients, although a single person often comes to seek information before he or she is emotionally involved and contemplating marriage. The information sought concerns the ability to have viable children in the case of the habitual aborter and/or to have "normal" children, especially where a child has already been born with "abnormalities." Using pedigree analysis and where necessary chromosome analysis, dermatoglyphics, and in some cases sex-chromatin analysis, along with other biochemical techniques, the genetic counselor arrives at a risk estimate for the prospective children of this couple or individual. The idea of risk estimate or probability is generally not well understood, and the counselor must be sure that the prediction and its meaning are properly understood. Educated and uneducated have incorrect notions of probability which lead them to believe that once an independent event occurs, such as the birth of an abnormal child, the chance that another such event will occur is far smaller than the individual chance. We all tend to forget that chance has no memory.

It is vitally important to elicit pedigree information properly and to be aware of the tensions which exist in a particular couple. As an example, if the husband is uncomfortable and seems anxious to lift blame from himself, you don't ask him about retardation in his family; rather, one might ask about levels of education or occupations of

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* Summary of presentation at the Physician-Clergy Symposium in Washington, D. C., May 3, 1968, sponsored by the Committee on Liaison with the Clergy of the Medical Society of the District of Columbia.

Opinions expressed in contributions to the Editorial Section are those of the writers and do not necessarily reflect the views of the Medical Society of the District of Columbia

Drug Dependence—Hallucinogens

Hallucinogens (LSD-25, mescaline and psilocybin—commonly known as LSD and acid): LSD-25 is a lysergic acid derivative. Mescaline is a chemical taken from peyote cactus. Psilocybin is synthesized from Mexican mushrooms.

Taken in tablet, capsule, ampul (hypodermic) form, or in saturated sugar cubes they produce hallucinations, exhilaration, or depression, and can lead to serious mental changes, psychotic manifestations, suicidal or homicidal tendencies.

Abusers may undergo complete personality changes, "see" smells, "hear" colors. They may try to fly or brush imaginary insects from their bodies, etc. Behavior is irrational. There is marked depersonalization.

Very small quantities of LSD may cause hallucinations lasting for days or repetitive psychotoxic episodes, which may recur months after injection. Permanence of mental derangement is still a moot question. Damage to chromosomes, and hence potentially to offspring, has been demonstrated.

brothers and sisters, etc. Many other methods can and should be used to respect the very difficult, and for many humiliating, task of studying their families for clues to present troubles. Often the party with the greater feeling of guilt or responsibility will be very quick to point out memory lapses in their opposite, saying with a smile, "Didn't your aunt have several miscarriages?" or something of the sort. These signs of tension and guilt should be considered in the total effort of the counselor to allow this coupl to make a reasonable, informed decision concerning their future reproduction.

It seems necessary to me that those who as a result of genetics counseling know what the best estimate of their risk is of having abnormal children must be encouraged to match this knowledge with their present marital situation, their need as individuals and as a family, their present and future health, financial position, and psychological stability. All of these factors should influence their decision. I always stress the need for an understanding of the feelings and sensitivity of both husband and wife in this matter. They must talk and realize what this information means to each individual; then by their love for each other they can work out a decision. It is easy to stress the fact that a couple who does this sort of communicating, even on one important level, has a much increased chance of having a good, meaningful marriage.

I encourage couples to get advice and help in making their decision, and the clergy of their own denomination may be very helpful in supporting them at such a difficult time. As for myself, as both geneticist and priest, I believe my dual role is advantageous. People facing such a momentous decision want to be assured of the counselor's interest in them as individuals and his commitment to a cultural heritage which goes beyond the present physical difficulty. I try not to use my position to influence the decision to be made, and as far as I know most genetics counselors adhere to this position. To the constant question, "What would you do if . . . ?" I refuse an answer except to insist that "only you as a couple can make a decision proper for yourselves."

Robert C. Baumiller, S.J., Ph.D.
Department of Obstetrics and Gynecology,
Georgetown University Hospital

How to Handle the Q & A*

For handling the Question and Answer period there are 10 important points. Remember that we have a Q & A in the first place because it:

1. Reinforces the main speech.
2. Increases the audience's knowledge.
3. Adds to the speaker's stature.
4. Provides valuable feedback information.

To achieve these goals, we strive for a Q & A that generates lively audience participation, provokes intelligent, stimulating questions, and leaves behind a residue of good feelings. These are the guidelines we should follow:

1. Announce it in advance. Let the program chairman know before the day of the meeting that you would like to have a Q & A. Arrange for the Q & A to be announced during your introduction.

2. Be prepared. Anticipate the type of questions you will be asked. Prepare printed reference materials. Arrange for a "plant" to be in the audience.

3. Get started smoothly. Avoid undue delay in beginning the Q & A. If questions are slow in

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* Editor's note: This is the summary, reprinted by permission, from a booklet by the same title, prepared by Robert Haakenson, Ph.D., of the Department of Public Relations, Smith Kline & French Laboratories, Philadelphia. The Executive Office of the Medical Society has on hand a number of copies of this booklet, delightfully illustrated, available upon written or telephone request (223-2230).

coming, relax. Recognize questioners with precision, and make sure that questions are heard and understood by all.

4. Analyze before answering. Spot the "loaded" question and point out its false premises while replying as best as possible. Divide complex questions into separate parts. Accept statements and other non-questions and respond to them just as to questions. Dismiss irrelevant questions.

5. Answer directly and back up your statements. Respond to all questions, even the "sticky" ones. Use "specifics" and documentation to support your arguments. Don't read the "heart" of your answer from your printed materials.

6. Stick to your specialty. Tackling questions outside your field is risky. Admit it when stuck: "I don't know." If you call on allies for help provide a graceful out to your invitees.

7. Be fair. Give everyone a chance. Don't let one person monopolize the Q & A: or trap you in an argument. Never take it upon yourself to "discipline" a questioner.

8. Keep Q & A moving. Establish a brisk rhythm to the Q & A; brief questions, brief answers, quick recognition of the next questioner.

9. End artistically. Don't drag out the session; stop while you're ahead. Anticipate the end, and close with a summary statement.

10. Follow up the Q & A. Make yourself available for an informal post-adjournment Q & A. Be relaxed, but discreet. This is a good time for developing future speaking engagements.

Now, are there any questions . . . ?

Sealtest
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Season's Best

Christmas Giving by Computer

With the computer taking over more and more of our everyday decisions it occurs to me that its finest hour might be in the realm of Christmas giving. What could be more appropriate than programming the whole family on the computer and letting the machine decide what to give.

For instance, starting with grandma and grandpa, we can go on to mother, father, wife, husband, brothers, sisters, uncles, aunts, cousins (not forgetting the children), listing for each one his/her age (approximately, of course), state of health and vigor, occupation, hobbies and interests, what he or she now has in the way of clothes, cars, furniture, jewelry, china, brica-b-rac, gadgets, et cetera. In the case of Baby Tim—what if anything is left of last year's toys, how long they lasted him, what changes have occurred in his size, shape, constructive abilities, destructive abilities, and so on. Friends and colleagues would require other data. To provide the information would be exhausting but not necessarily impossible and could be done in armchair comfort.

Then feed into the computer the amount you know you will but can't afford to spend and sit back for the results. In this affluent society the computer may simply disintegrate, proving that the human being is the more durable machine, or it may propose a $25 Christmas card for each person on the list. But what a lot of headaches and explanations that would take care of.

A. L. E.

Grandpa's Blessing*

We children waited by each breakfast plate;
Impatiently, we drummed the silverware.
Grandpa loved the summer's sleeping late;
Breakfast would not begin 'til he was there.

And often he had only just begun,
Just shaved, washed, and finished dressing,
(A doctor learns to sleep as best he can)
When he sat down to give the daily blessing.

On this day, awaking had been quite a fight!
As he took his seat, our chairs were squeaking.
With towheads bowed and eyes closed tight,
We heard the blessing start, "Doctor Macatee
speaking"—
And though we snickered with our heads still bent,
We kids knew then what "In Thy service" meant.

Virginia McFee

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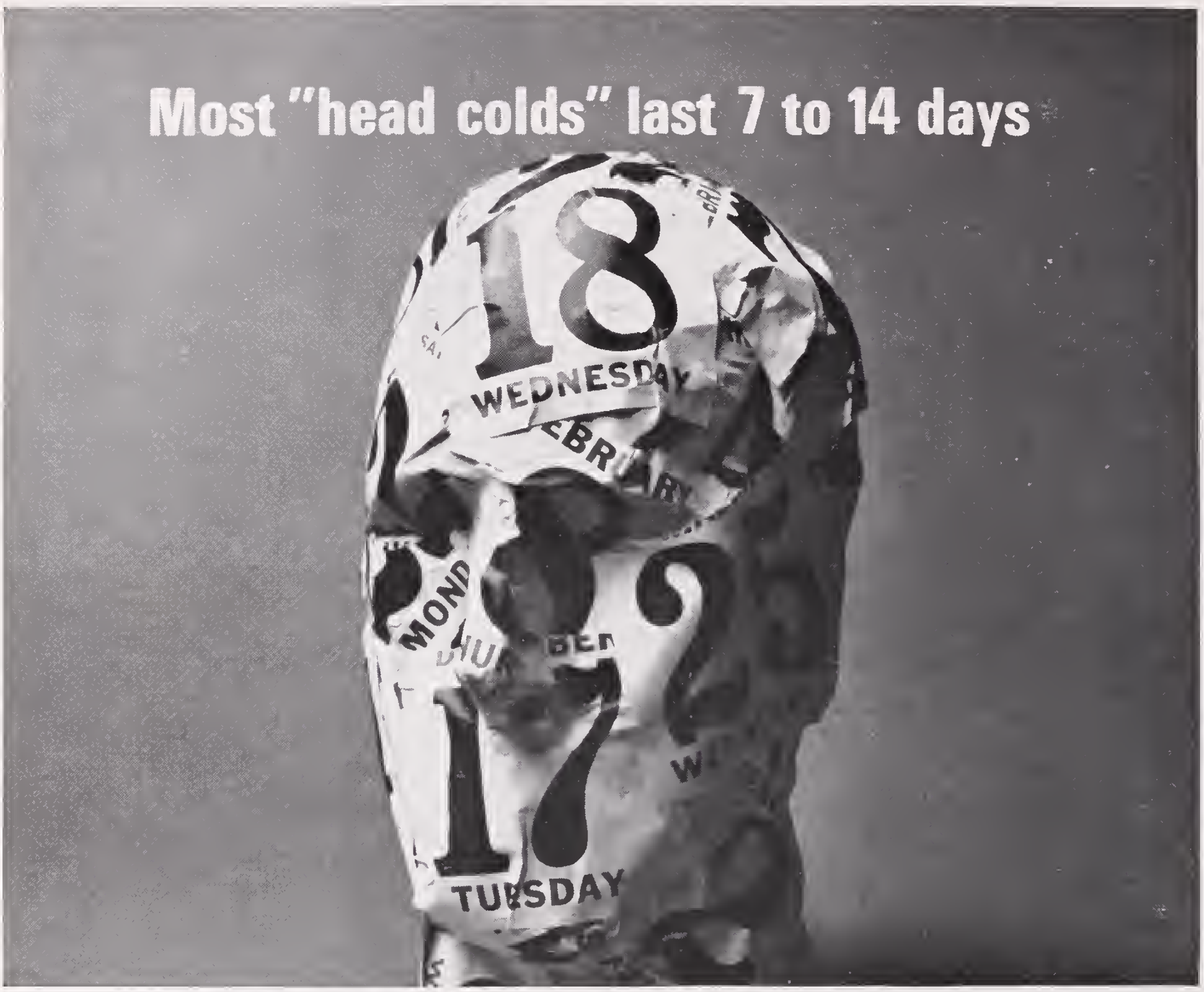
*Submitted to the Medical Annals by Mrs. Thomas McFee, a granddaughter of the late Dr. Henry C. Macatee.



In the meantime...Ornade®

Prompt relief from nasal congestion and hypersecretion due to colds.

Before prescribing, see complete prescribing information in SK&F literature or *PDR*.
Contraindications: Glaucoma, prostatic hypertrophy, stenosing peptic ulcer, pyloroduodenal or bladder neck obstruction.
Precautions: Use cautiously in the presence of hypertension, hyperthyroidism, coronary artery disease, warn vehicle or machine operators of possible drowsiness.
Usage in Pregnancy: Use in pregnancy, nursing mothers and women who might bear children only when potential benefits have been weighed against possible hazards.
Note: The iodine in isopropamide iodide may alter PBI test results and will suppress I^{131} uptake; discontinue 'Ornade' one week before these tests.
Adverse Reactions: Drowsiness, excessive dryness of nose, throat or mouth; nervousness; insomnia. Other known possible adverse reactions of the individual ingredients: nausea, vomiting, diarrhea, rash, dizziness, fatigue, tightness of chest, abdominal pain, irritability, tachycardia, headache, incoordination, tremor, difficulty in urination. Thrombocytopenia, leukopenia and convulsions have been reported.
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The New AMA Division of Public Affairs

As of September 23, 1968, the American Medical Association established a new Division of Public Affairs. It amalgamates the field staffs of the AMA and AMPAC (American Medical Political Action Committee), assumes the functions of the Division of Field Service, and takes on broader responsibilities in the public affairs area. These include a variety of community, civic and governmental activities, all vitally important to the medical profession and its ability to provide high quality health care for the American people.

The new Division of Public Affairs will be charged with implementing AMA policy that involves the necessary relationships between medicine and Government and with responsibility for conducting many of the education and research programs formerly carried out by AMPAC.

Mr. Joe D. Miller, former Executive Director of AMPAC, has been named Director of the new Division. Mr. Aubrey Gates, former Director of the Division of Field Service, has been appointed Executive Assistant in the office of the Executive Vice President of the AMA. Mr. David W. Powers, former Assistant Director of AMPAC, has been named by the AMPAC Board of Directors as Acting Executive Director of AMPAC.

AMPAC will remain separate from the AMA and be governed as before by its own Board of Directors. It will continue to function as the political action arm of organized medicine.

The Thinking Behind the AMA Decision

In recent years the conditions under which medicine is practiced have changed greatly. So too has the capability of the medical profession to treat patients.

The structure and functions of the AMA must accordingly be revised to direct these changes in ways that best serve medicine and the public.

Medicine and its practice depend increasingly on public attitudes. Public attitudes in turn affect Government. And Government itself has within its power the ability to alter — for better or worse — the way in which medicine is practiced. At stake is the effectiveness of the health care available to 200 million Americans.

The interrelationship between the medical profession, the public, and Government is increasingly intimate as a result of some relatively recent developments:

1. The so-called scientific explosion, which has revolutionized medical science and technique and vastly increased the value of what medicine has to offer.
2. Public awareness of this, leading to a surging demand for the best that medicine has to offer.
3. Growing affluence, which enables a sizable portion of the public to expect it should have the best of everything.
4. Health insurance, increasingly widespread, which brings quality care within the means of millions and increases the demand for it.
5. The influence of labor unions, liberals in both political parties, and intellectuals of media and Government in pressing for social benefits for large segments of our society.
6. Public acceptance of Government as a legitimate social and economic planner.

These and other factors have altered the environment in which medicine is practiced, health care is sought, and Government seeks to act.

Meanwhile, medicine's problems are complex and pressing: the shortage of physicians and other members of the health team in relation to the multiplied demand; the gap between demand for hospital and nursing home services and their availability; the rising costs of health care. These and other problems must ultimately be solved, either by the private sector or—by default—Government.

The critical element in medicine's search for solutions is time. Given enough time, most of the answers can be found by the private sector. Given insufficient time, Government is likely to act precipitately in response to public pressure.

For the foreseeable future, changes in medical science and practice will affect the environment in which they are made. And at the same time that environment will influence the manner in which medical science and practice function.

The AMA, as the organization that unifies the Nation's physicians, must prepare to meet present and future health needs in the United States, direct the present trends to the degree legally feasible, and argue its case persuasively with the Government and the public.

Thus, no aspect of the AMA's planning, operation or activity is independent of what the AMA is able to accomplish in public affairs. This is true even of the scientific and medical areas in which the AMA functions so competently; the ability to perform capably in these areas requires the freedom in which to do it.

A new approach is therefore required, along with a new organizational structure with which to execute it. The creation of a new Division of Public Affairs is a first step in that direction. The move is practical and logical. It will result in better staff coördination, administrative efficiency, and economical operation. When fully operative the Division of Public Affairs will be capable not only of providing better legislative and socioeconomic services to the constituent and component medical societies but of coördinating them better from the management to the field levels.

The decision to create the new Division responds to a resolution introduced by the Illinois delegation and referred to the Board of Trustees by the House of Delegates at the June, 1968 meeting of the AMA. The resolution called for the development of a public affairs program within the AMA.

The Responsibilities of the Public Affairs Division

One definition of public affairs is "a systematic effort to safeguard an organization's rights and fulfill its responsibilities to the community while encouraging individual members of the organization to safeguard their own rights and fulfill their own responsibilities."

Applying that definition, then, the Division of Public Affairs will systematically undertake programs to safeguard the rights of freely practiced medicine, to assist the AMA in its efforts to discharge its responsibilities to the profession and the Nation, and to encourage individual physicians and their wives to safeguard their own rights while fulfilling their own responsibilities as members of a free society.

The goals of the Division are:

1. To help inform the profession, public and legislators of the problems facing medicine.
2. To foster a better understanding of Government on the part of physicians and their wives and to stimulate their activity in civic and political affairs.
3. To help medicine amplify its voice in community, state and national affairs.
4. To disseminate information of interest to legislators and maintain a practical legislative liaison.
5. To encourage physicians and their wives to join the PAC movement and to become active in the political party of their choice.
6. To serve the component and constituent medical societies effectively and frequently with legislative and political information.
7. To provide nonpartisan political education programs for individuals and medical societies.
8. To undertake such objective, educational research as may be helpful.
9. To furnish close liaison between the AMA and the component and constituent societies to foster communication on public affairs.
10. To maintain contact and improve coördination with other public affairs movements of a nonmedical nature.

In the performance of these functions, the Division of Public Affairs will work closely with the other divisions of the AMA and with appropriate Councils, and will routinely maintain liaison with AMPAC and State PACs.

The Division will be responsible for the legislative representation of the AMA with the Congress and for routine liaison with the Executive Departments and agencies of the Federal Government.

The Role of AMPAC

AMPAC will maintain its individual entity as an organization separate from the AMA and governed by its own Board of Directors. As in the past, it will function as the political arm of medicine.

AMPAC will continue to conduct certain research programs, publish *Political Stethoscope*, maintain a lending library of films and political books, and conduct selected programs of political education.

Its staff will maintain up-to-date files on the political situation—state by state and district by district. This will be available to the Review Committee of the AMPAC Board routinely and will guide the Committee in its decisions involving candidate support.

AMPAC will continue to serve the individual State PACs in matters involving political action, the exchange of information, the transmittal of membership dollars and political allocations, etc. But with the establishment of the new Division of Public Affairs coördination of AMPAC activities with those touching on legislation and regulation will be closer and more effective.

As AMA salaried employees the field staff of the Division of Public Affairs will not, of course, engage in political activities in which the support of candidates for nomination or election to public office are involved. Such activities will continue exclusively as AMPAC functions.

Conclusion

Some details of this reorganization have not yet been worked out. Others may not be finally resolved until a management study, recently initiated, develops its final recommendations for the administration of the AMA.

AMPAC anticipates no transitional problems arising from the reorganization that will in any way diminish the effectiveness of its ability to support candidates.

As for the future, not only does the reorganization enhance the role of political action in medicine's public affairs complex, it also reinforces the political education effort with the full resources of medicine.

In summary, then, the reorganization produces immediate benefits to both the AMA and AMPAC. Those benefits will be multiplied in the years ahead as improved staff coördination, greater administrative efficiency, better liaison and enhanced services to the component and constituent societies pay dividends to organized medicine at every level of its participation in public affairs.

HEALTH DEPARTMENT NOTES

The Behavioral Scientist

The Health Education and Information Division of the D. C. Department of Public Health has the responsibility of promoting the Department's programs both through direct contact with consumers of health services and through the use of mass news media.

Although many accept good health as important there are pockets of resistance. Finding the reasons why a certain program succeeds or fails often becomes the responsibility of the staff of the Behavioral Sciences Section of the Health Education and Information Division.

The first concern of the Behavioral Sciences Section is public acceptance of a health program, particularly when a new approach is used in offering services to the public. Clearly the public, as consumers of health services, is the most vital factor in planning any program. If a particular project falls short of its goal, changes must be made before another attempt is considered.

When a new health program is initiated the behavioral scientists explore field conditions to determine the reasons for success or failure. The attitudes of potential users of health services are interpreted. Findings are translated into recommendations which may serve as guidelines for administrative or field actions.

Results of studies and recommendations are transferred to health educator program chiefs and aides through group or individual briefings, all aimed at furthering acceptance and support for Health Department programs.

During the past year there were a number of health programs studied and evaluated by our behavioral scientists. One such effort revolved around teaching young children in the Head Start program good health and grooming habits. A clown called "Baffo" was used to help get the messages across in words, songs, and visual aids. After more than 4,500 children witnessed the show, a study and evaluation was made through questions asked of both the youngsters and their teachers.

Another study was made in connection with the Health Department's Birth Control program. Results were mixed. Many of the individuals approached thought that birth control was good for their friends and neighbors but not for themselves. The extent of this negativism pointed to the need for a comprehensive educational program to promote a more favorable attitude toward the subject.

A third project was to determine the level of familiarity with selected services of the D. C. Department of Public Health. The study, made at D. C. General Hospital, inquired into how many individuals utilized Department services once they were known and whether or not Health Department educational materials they read were properly understood. The fact that the persons interviewed were already utilizing a health facility at the time of the interview did not put the questions on a fair basis of operation. However, there were some interesting observations. Greatest familiarity existed with such popular facilities as chest X-rays, family planning, Alcoholic Rehabilitation Clinics, and the Maternal and Infant Care Clinics. How they heard about these also was revealing. Most said they heard about these services through word of mouth and television; few bothered to read newspaper stories on the same subjects.

Whatever the program may be, it is important that the behavioral scientists study patient needs and attitudes. Through applied research they can determine how Health Department programs might be made more effective through design or modification.

Murray Grant, M.D., D.P.H.
D. C. Director of Public Health

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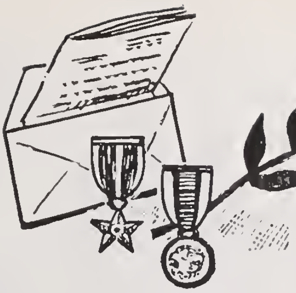

News & Personals

Summary of Executive Board Actions

Proceedings of October 21 Meeting

Dinner for the Board members was served in the Exhibit Hall of the Medical Society. Dr. Edward W. Nicklas, Chairman, presided at the meeting, which was attended by Drs. William S. McCune, Frank S. Bacon, Gudrun Augustin-Rueckert, Paul R. Wilner, Robert J. Coffey, Henry D. Ecker, Raymond T. Holden, John J. Hughes, and Mr. Thomas Sadler, Secretary. Present by invitation was Dr. Arthur E. Wentz, Program Coördinator, Metropolitan Washington Regional Medical Program. The meeting was called to order by Dr. Nicklas at 7:20 p.m.

The minutes of the September 23, 1968 Board meeting were approved as submitted.

Committee Meetings: The Secretary reported the following committees had met since the last meeting of the Board:

October 1	Committee on Relative Value Study
2	Fee Review Committee
3	Committee on Medical Aspects of Sports
4	Committee on Blood Banks
8	Board of Credentials
10	Physicians' Placement Committee
11	Public Welfare Committee
15	Committee on Medicolegal Problems

Board of Credentials: On recommendation of the Board of Credentials the following applicants were elected to membership:

Active

Charles Rocco Tartaglia, M.D. Office: 3800 Reservoir Road, N.W. Born in Washington, D. C. Medical degree: Georgetown University, 1962. Specialty: Psychiatry.

Karl Wipplinger, M.D. Office: George Washington University Medical Center. Born in Steyr, Austria. Medical degree: University of Vienna, Vienna, Austria, 1953. Specialty: Internal Medicine.

Associate

Henry Smither Gering, M.D. Office: 4400 Stamp Road, S.E., Marlow Heights, Md. Born in Washington, D. C. Medical degree: George Washington University, 1961. Specialty: Obstetrics and Gynecology.

Ciriaco Morales Lorenzo, M.D. Office: 50 Irving Street, N.W. Born in Gattaran, Cagayan, The Philippines. Medical degree: University of Santo Tomas, Manila, The Philippines, 1956. Specialty: Anesthesiology.

Samuel F. Mazella, M.D. Office: 2704 Riveria Street, Marlow Heights, Md. Born in Passaic, N. J. Medical degree: George Washington University, 1961. Specialty: General Surgery.

Resident-Intern

John J. Calarco, M.D. Office: St. Elizabeths Hospital. Born in New York City. Medical degree: Georgetown University, 1938. Specialty: Pathology.

Approval was given to the following changes in membership status:

Transfers

Active to Associate: Dale C. Cameron and Stanley H. Steinberg

Resignation

Associate: Joseph M. O'Neil

The dues of Dr. Howard M. Skolnick were remitted because of illness.

Regional Medical Program: Dr. Arthur E. Wentz, Program Coördinator, presented a progress report on the Program which was divided into 2 parts, professional and administrative. The Board members were in full agreement that Dr. Wentz should come to them at any time for guidance and assistance and that, when necessary, special board meetings would be held to accommodate him. It was also agreed that for the time being no action be taken to appoint a Society representative for RMP.

New Consulting Editor: Approval was given to a recommendation from Dr. Wallace M. Yater, Editor, that Colonel Robert H. Moser, MC, USA, succeed Colonel Loren F. Parmley, MC, USA, as Consulting Editor to the Medical Annals, repre-

COMMUNITY HEALTH WEEK—the good start was sustained

Community Health Week, observed in the District of Columbia, October 20–25, was promoted by the Medical Society of the District of Columbia with free health tests to all comers to its building during the first 4 days. Coöperating were the D. C. Department of Public Health, the Washington Hearing Society, and the Prevention of Blindness Society of Metropolitan Washington.

(Continued on opposite page)

senting Walter Reed General Hospital. Colonel Moser, like his predecessor, is Chief of the Department of Medicine at Walter Reed.

Proposed PR Program: Dr. McCune outlined a proposed public relations program which will actually go into effect on a trial basis prior to and during the ASSEMBLY. In substance, the program calls for a Society employee to handle all PR activities. During the first 6 to 8 months of the program the employee will be under the guidance of a PR consultant.

Dr. McCune also announced the cancellation of the proposed November 7 cocktail party for TV, radio, and press officials. Instead, he will approach in person local newspaper editors.

Proposed Travel Club: The proposal of United Travel Service to establish a Medical Society Travel Club was approved in principle.

Establishment of New Division of Public Affairs within AMA: Information pertinent to this item had been mailed to each Board member. (Following the meeting of the Board an informative article covering the background of the establishment of the Division was received in the Executive Offices and is published in this issue of the ANNALS, beginning on page 671.)

Other Business

1. Motion was made and carried that the Medical Society go on record as approving helicopter ambulance service in principle and that the Health Foundation be requested to lend financial assistance where possible.

2. Dr. McCune reported on a recent meeting with Attorneys Barse and Laskey who advised that the Society proceed as planned with the proposed revisions to the Licensure Law.

3. The Board authorized Dr. Harry Zehner, Jr., Vice Chairman of the Emergency Medical Service Committee, to attend an AMA-sponsored Conference on Disaster Medical Care in Albuquerque, New Mexico, November 15 and 16, 1968.

There being no further business, the meeting was adjourned at 9:55 p.m.

GENERAL NEWS

Two World Congresses, representing neurologists and neuosurgeons, have issued a **call for abstracts of scientific material** to be presented at a joint meeting to be held, September 20–27, 1969 at the New York Hilton Hotel in New York City.

Deadline for submitting abstracts for the Ninth International Congress of Neurology and the Fourth International Congress of Neurological Surgery is January 15, 1969. The abstracts, which are not to exceed 200 words, will be considered for major theme papers, free communications, motion picture presentations, and scientific exhibits.

Official abstract form and other information on the Congresses may be obtained from the World Congresses of Neurological Sciences, Suite 417, 420 Lexington Avenue, New York, N. Y. 10017.

Mr. Joe D. Miller, Director of the new Public Affairs Division of the American Medical Association (see page 671 of this issue), states that field activities of the Division can best be handled by representatives headquartered within the area of their own assignment. The AMA therefore plans 12 field offices staffed by 12 representatives of the Public Affairs Division. The District of Columbia will be assigned to **Mr. Chad Combs,** former AMPAC Field Representative, whose office will be located in Lemoyne, Pennsylvania. He will also serve the state medical associations of Maryland, Pennsylvania and Virginia.

The American Association of Blood Banks has established a **scholarship awards program**

(Continued from opposite page)

The Week opened with a Physicians' Jog, starting at the Washington Monument and concluding at the Medical Society's Building. Dr. Warren D. Brill (top, left end) chairmaned the event with much assistance from Dr. Nelson G. Goodman (center of standees) and Dr. Donald D. Haut (kneeling, front right), and many others.

Center row, left: Patient from Columbia Lighthouse for the Blind gets a hearing test, with assurance from her little friend; at right a student from Gallaudet College takes her test for visual acuity.

Lower left: Student from Gallaudet College receives a blood test from Milton Anderson, Chief Medical Technologist for the D. C. Health Department, while another student awaits her turn. Lower right: Bonny Falk, of the Annals staff, is given an electrocardiogram by Maryuella Quarles, R.T., also of the D. C. Health Department.

Chest X-rays were taken in the Health Department's mobile testing unit stationed outside the Medical Society's Building.

A total of 1,165 persons received free tests, which, in addition to those mentioned, included tests for glaucoma and diabetes; 154 persons appeared for testing on the final day.

that will enable medical technologists to acquire certification in blood banking. The certification will be conferred jointly by the AABB Committee on Education and the Registry of Medical Technologists of the American Society of Clinical Pathologists.

Interested individuals and companies may sponsor scholarship awards which will take the form of grants to schools approved by the AABB for education in blood banking. The medical director of each approved school will then select the technologist most deserving of the scholarship award.

In establishing the program, the AABB cited the need for more trained technologists to meet the standards of excellence required in the rapidly expanding field of blood banking.

The sixth revision of the **AMA Directory of National Voluntary Health Organizations** is now available. Produced by the Council on Voluntary Health Agencies, it describes the purpose, organizational pattern, financing, and program of the various national voluntary health agencies and other organizations of medical interest. Listing of an agency in the Directory is for informational purposes and implies neither approval nor disapproval by the AMA. A single copy of the Directory may be secured by addressing the AMA Council on Voluntary Health Agencies, 535 North Dearborn Street, Chicago, Ill. 60610.

Georgetown University School of Medicine is the recipient of a $50,000 grant from Gulf Oil Corporation to be used to plan a model health program at Reston, Va. Dr. Patrick J. Doyle, Chairman of the Department of Community Medicine and International Health, will direct the pilot program, which is aimed at preventing disease through health education.

Mr. Richard M. Loughery, Administrator of Washington Hospital Center, was recently elected to the Council of Regents of the American College of Hospital Administrators. Each Regent is elected by the members in his own state or province. Mr. Loughery, a Fellow of the College, will represent the District of Columbia during a 3-year term.

PERSONALS

Dr. Ernest A. Gould has been named full-time Chairman of the Washington Hospital Center's Department of Surgery. In this post he will be responsible for the overall surgical training program for interns and residents and the coördination of this program with the surgical staff. He is senior attending surgeon at the Center and past Chairman of the Department of General Surgery.

Dr. Gould, new Chairman, Department of Surgery, Washington Hospital Center

A graduate of George Washington University School of Medicine, Dr. Gould is on the faculty as Associate Clinical Professor of Surgery and Associate in Surgery at the University Hospital and at District of Columbia General Hospital. He is a diplomate of the American Board of Surgery, a Fellow of the American College of Surgeons, and

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Treasurer of the American Thyroid Association, as well as a member of numerous other scientific organizations.

Dr. A. Frederick North, Jr. has been appointed Associate Director of Children's Hospital for Ambulatory Patient Services, a newly created administrative position. He will be responsible for program development and administration of all services to ambulatory patients, which include the Hospital's Diagnostic and Treatment Center, Emergency Clinic, and Comprehensive Health Care Program. He will also serve as Associate Professor of Pediatrics at George Washington University School of Medicine and as Senior Attending Pediatrician at Children's.

Dr. North, a graduate of Yale University School of Medicine, came to Children's from the U. S. Office of Economic Opportunity where he served as senior pediatrician since 1966 of Project Head Start. Prior to that he lived in Rochester, New York, where he was Director of the Pediatric Clinic at the Strong Memorial Hospital; Director, Pediatric Section, Arthritis Clinical Study Center of the University of Rochester; and Assistant Professor of Pediatrics at the University of Rochester.

Dr. Roland B. Scott was in Chicago, October 19–24, attending the annual meeting of the American Academy of Pediatrics. During the meeting he served as an examiner for the American Board of Pediatrics for candidates who were taking examinations for certification in pediatric allergy. Dr. Scott and Dr. M. Nick Sherry both presented papers at the meeting.

Dr. Wallace M. Yater was honor guest at the luncheon, October 12, of the D. C.-Maryland 20th Annual Regional Meeting of the American College of Physicians, held at Georgetown University. Dr. Yater has served the College as Governor for the District of Columbia (1933–1949), Regent (1949–1956), and Secretary-General (1956–1968). He is an alumnus and former Professor and Chairman of the Department of Medicine at Georgetown University School of Medicine.

Dr. Thomas W. Mattingly, presently ACP Governor for the District of Columbia, presided at the luncheon and briefly outlined the many contributions which Dr. Yater has made to Georgetown and to the College of Physicians during 35 years as an officer of the latter. Dr. John C. Rose, Dean of Georgetown University School of Medicine, and Dr. Mattingly jointly presented Dr. Yater with a set of his collected papers, beautifully bound in 2 volumes, as a token of appreciation for his services and devotion to these 2 institutions. A second set will be placed in the Medical Library at Georgetown.

DR. WALLACE M. YATER, honored guest at Regional Meeting of the American College of Physicians, accepts 2 bound volumes of his own writings from Dr. Rose, Dean of Medicine at Georgetown University.

Dr. William Tenenblatt was recently elected President, and **Dr. George J. Ellis, Jr.**, Secretary-Treasurer, of the Montgomery County (Maryland) Gynecological Society. Dr. Tenenblatt practices in Silver Spring and Dr. Ellis in Rockville, Md. Both are Associate members of the D. C. Medical Society.

The largest number of surgeons in the history of the American College of Surgeons were inducted, October 17, as new Fellows of the College during its Annual Clinical Congress. Physicians from the District of Columbia who were inducted into fellowship at that meeting were: **Drs. David H. Bachhuber, Elizabeth A. Crisp, N. Thomas Debevoise, Oscar S. DePriest III, Philip J. Ferris, John L. Fox, Leroy Jones, Alfred J. Luessenhop, William S. Lyons, Robert C. Nothwanger, Jerome L. Sandler, Robert L. Simmons, and Olden E. Stinson.** All are Active members of the Medical Society except Dr. Nothwanger, who is an Associate member.

DOCPAC BRIEFS

or

A Friend in the White House

THE ELECTION IS OVER and medicine again has a friend in the White House! President-elect Richard M. Nixon is dedicated to the continuation of the practice of medicine with as much freedom from Government interference as possible, and with freedom of choice for the patient.

However, we must not let down our guard. The liberals and the "bleeding hearts" will again try to put medicine in "socialized form" or at least under the control of the multiple bureaucracies in Washington.

And again, beginning in 1969, DOCPAC (your District of Columbia Political Action Committee) will be soliciting funds from you to be placed with both Republicans and Democrats who are members of key committees pertaining to health legislation of both the Senate and the House. These monies will help them get reëlected.

You will notice that we now have joint billing. With your dues for the D. C. Medical Society there is a statement asking your voluntary help for DOCPAC. We hope that all of the physicians of the District of Columbia Medical Society will become more politically conscious. Your aid in keeping medicine as free from Government intervention as possible is very much needed.

WILLIAM H. COOPER, M.D.
Chairman, D. C. Political Action Committee

Patients Needed at Clinical Center

THE COÖPERATION OF PHYSICIANS is requested in the referral of patients with hyperbilirubinemia for studies of bilirubin metabolism being conducted by the Metabolism Branch of the National Cancer Institute at the Clinical Center, National Institutes of Health, Bethesda, Md.

Of particular interest are patients with chronic or intermittent unconjugated hyperbilirubinemia in whom the diagnosis of hemolysis, Gilbert's syndrome, or a post-hepatitic syndrome is suggested. Patients with conjugated or mixed hyperbilirubinemia (e.g., Rotor's syndrome or Dubin-Johnson syndrome), in whom obvious obstructive or active inflammatory disease has been ruled out, are also needed.

Selected patients will be admitted to the Clinical Center as inpatients for a minimum period of 2 weeks. Isotopic bilirubin clearance and red blood cell life-span studies, as well as dye clearance studies, will be performed. Liver biopsy may be performed in selected situations. Upon completion of their studies patients will be returned to the care of the referring physician, who will receive a summary of findings.

Physicians interested in having their patients considered for admission to these studies may write or telephone Dr. Joseph R. Bloomer, Clinical Center, Room 4-N-117, National Institutes of Health, Bethesda, Md. 20014; telephone (301) 656-4000, Ext. 65955.

LETTERS TO THE EDITOR

Disposal of Drug Samples

I should like to bring to the attention of all members of the Medical Society that an increasing number of adolescents report that they are picking up discarded drug samples from the offices of physicians who are throwing samples received in the mail into trash baskets. Adolescents are finding such samples when they rummage through the trash bins.

Most of the drugs shown to me by adolescents have been tranquilizers or antihistamines. The packages are labeled "Professional Samples," leaving no doubt of the source of supply. Some of the samples have been drugs with addictive properties.

Because these adolescents are unsophisticated and will try any drug to see the effects it will produce, I advise all physicians to be very careful about discarding their samples. I would recommend either flushing the drugs down the toilet or separating them into smaller, unidentifiable packages before throwing them into the trash baskets.

I believe that we physicians may be unwittingly compounding the problem of drug usage in youngsters. As potential sources of supply we should move to exercise more control over our disposition of unwanted pharmaceutical samples.

EDWARD H. WEISS, M.D.

In the complex picture of moderate to severe anxiety...

there is a new reason for prescribing Mellaril (Thioridazine HCl)

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Long recognized for its usefulness in the treatment of moderate to severe anxiety, Mellaril is now also known to be effective against mixed anxiety-depression.

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Before prescribing or administering, see Sandoz literature for full product information, including adverse reactions reported with phenothiazines. The following is a brief precautionary statement.
Contraindications: Severe central nervous system depression, comatose states from any cause, hypertensive or hypotensive heart disease of extreme degree.
Warnings: Administer cautiously to patients who have previously exhibited a hypersensitivity reaction (e.g., blood dyscrasias, jaundice) to phenothiazines. Phenothiazines are capable of potentiating central nervous system depressants (e.g., anesthetics, opiates, alcohol, etc.) as well as atropine and phosphorus insecticides. During pregnancy, administer only when necessary.
Precautions: There have been infrequent reports of leukopenia and/or agranulocytosis and convulsive seizures. In epileptic patients, anticonvulsant medication should also be maintained. Pigmentary retinopathy may be avoided by remaining within the recommended limits of dosage. Administer cautiously to patients participating in activities requiring complete mental alertness (e.g., driving). Orthostatic hypotension is more common in females than in males. Do not use epinephrine in treating drug-induced hypotension. Daily doses in excess of 300 mg. should be used only in severe neuropsychiatric conditions.
Adverse Reactions: *Central Nervous System*—Drowsiness, especially with large doses, early in treatment; infrequently, pseudoparkinsonism and other extrapyramidal symptoms; nocturnal confusion, hyperactivity, lethargy, psychotic reactions, restlessness, and headache. *Autonomic Nervous System*—Dryness of mouth, blurred vision, constipation, nausea, vomiting, diarrhea, nasal stuffiness, and pallor. *Endocrine System*—Galactorrhea, breast engorgement, amenorrhea, inhibition of ejaculation, and peripheral edema. *Skin*—Dermatitis and skin eruptions of the urticarial type, photosensitivity. *Cardiovascular System*—Changes in the terminal portion of the electrocardiogram have been observed in some patients receiving the phenothiazine tranquilizers, including Mellaril (thioridazine hydrochloride). While there is no evidence at present that these changes are in any way precursors of any significant disturbance of cardiac rhythm, several sudden and unexpected deaths apparently due to cardiac arrest have occurred in patients previously showing electrocardiographic changes. The use of periodic electrocardiograms has been proposed but would appear to be of questionable value as a predictive device. *Other*—A single case described as parotid swelling.

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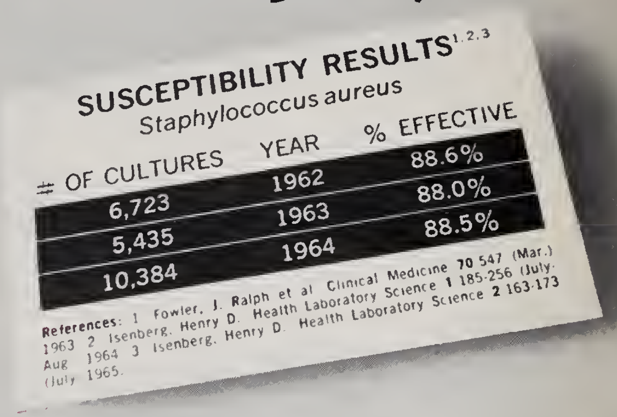

SUSCEPTIBILITY RESULTS[1,2,3]
Staphylococcus aureus

♯ OF CULTURES	YEAR	% EFFECTIVE
6,723	1962	88.6%
5,435	1963	88.0%
10,384	1964	88.5%

References: 1 Fowler, J. Ralph et al Clinical Medicine **70** 547 (Mar.) 1963 2 Isenberg, Henry D. Health Laboratory Science **1** 185-256 (July-Aug 1964 3 Isenberg, Henry D. Health Laboratory Science **2** 163-173 (July 1965.

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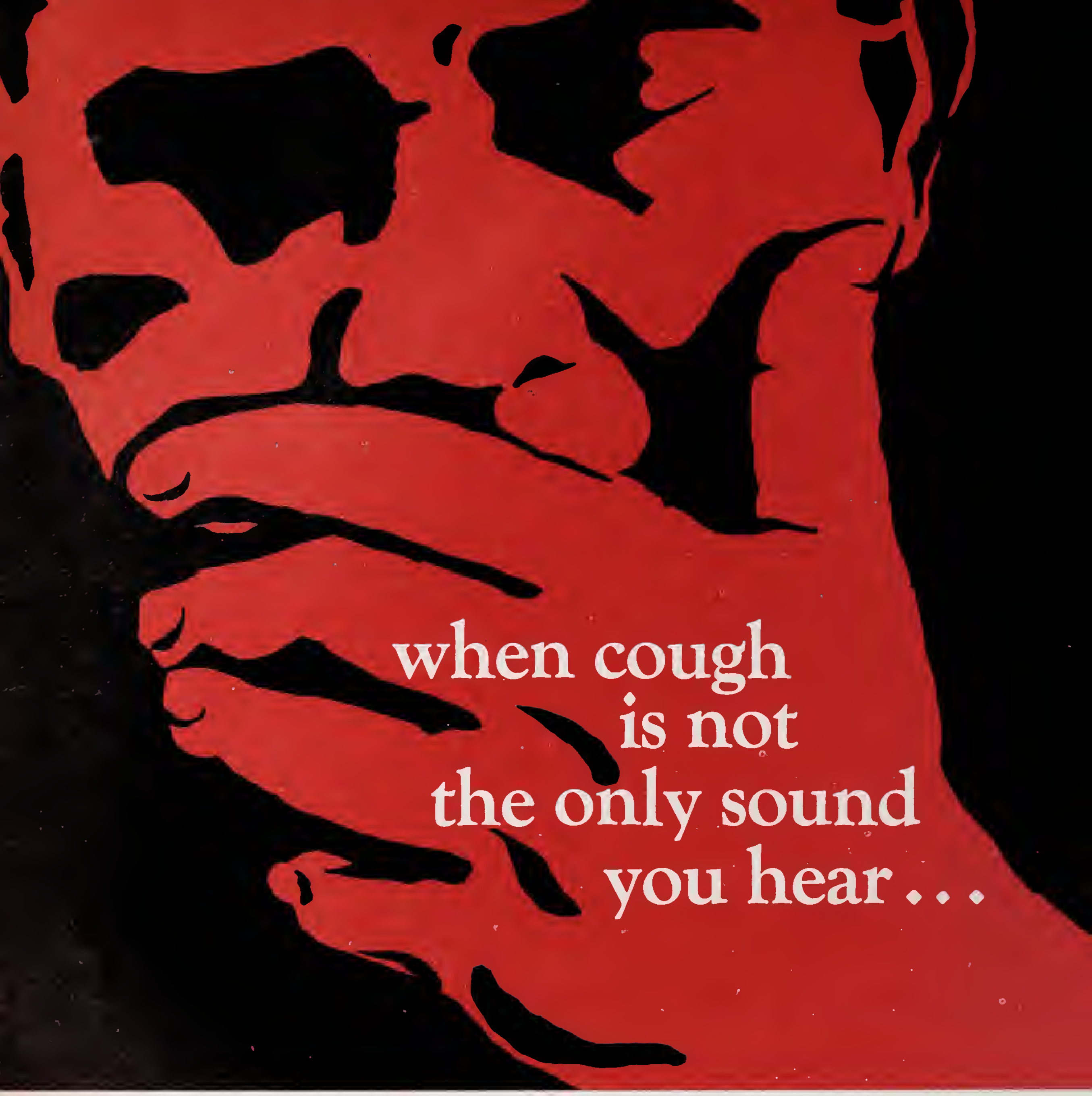

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OBITUARIES

Joseph Kreiselman, M.D.

(1895–1968)

Dr. Joseph Kreiselman, anesthesiologist in Washington for many years, died in Fort Lauderdale, Fla., October 21, 1968. He had been ill for many years.

A native of McKeesport, Pa., Dr. Kreiselman was born, May 1, 1895. He received his medical degree from George Washington University School of Medicine in 1919 and interned at the University Hospital. He was a resident physician at Columbia Hospital for Women in 1920.

Dr. Kreiselman is famous for the development of the Kreiselman Resuscitator used in the treatment of asphyxia of the newborn infant and now standard equipment in hospital delivery rooms. During World War II, as a consultant in anesthesiology to the Surgeon General of the Army, he introduced the bellows resuscitator which operates like a pair of hand bellows and uses room air. It was used in combat units during World War II.

Dr. Kreiselman first joined the faculty of George Washington University in 1927 as an instructor in anesthesiology. After World War II he returned to George Washington, where he taught until 1948. At that time he was Clinical Professor of Anesthesiology. For many years he was Chief of Anesthesiology at Emergency Hospital. He also practiced anesthesiology at Columbia, Garfield, and George Washington University Hospitals from 1920 to 1948. About 1953 he moved with his wife to Florida.

He was elected to Active membership in the Medical Society of the District of Columbia in 1925 and became a Life member in 1965. He was a member of the American Medical Association, the Hippocrates-Galen Medical Society, and the Washington Academy of Surgery. A diplomate of the American Board of Anesthesiology, he was a member of the American Society of Anesthesiologists and a founding member of the D. C. Society of Anesthesiologists. He contributed many articles to the literature, including several on resuscitation of the newborn.

Surviving him are his wife, Mrs. Molvine Kreiselman, of 1920 South Ocean Drive, Fort Lauderdale, Fla., and 2 daughters, Mrs. Josephine Spear, of Pompano, Fla., and Mrs. Marian Slater, of New York City. He also leaves 3 sisters, Mrs. Lee Jaffee, of Washington; Mrs. Gertrude Miller, of Cleveland; and Mrs. Helen Lippert, of Norwalk, Ohio, and a brother, Charles Kreiselman, of New York City.

Crenshaw Douglas Briggs, M.D.

(1904–1968)

Dr. Crenshaw D. Briggs, well known Washington surgeon, died, October 5, 1968 at the Washington Hospital Center after a long illness. He had been associated with Dr. Harold H. Hawfield in offices at 2001 Eye Street, N.W.

A native of Richmond, Virginia, Dr. Briggs was born, January 11, 1904. He received his Bachelor of Science degree from Virginia Military Institute in 1924 and his medical degree from Johns Hopkins University in 1928. He interned at Johns Hopkins University Hospital and continued his training there in surgery as assistant resident from 1929 to 1934 and as resident physician from 1934 to 1935.

During World War II he served as Lieutenant Colonel in the U.S. Army Medical Corps. He was Chief of Surgery at Finney General Hospital in Thomasville, Ga. and held a similar position at a hospital on a Tinian Island base in the Pacific.

In 1936, Dr. Briggs began his practice in Washington which was interrupted by his war service. He was Chief of Surgery at Emergency Hospital when it merged into the Washington Hospital Center in 1958, and Dr. Briggs was the first to head the Department of General Surgery at the Center, continuing after 1959 as senior advisory surgeon until his retirement last year. He also served as senior attending surgeon at Children's Hospital. He taught surgery at Johns Hopkins University and George Washington University Schools of Medicine.

Dr. Briggs was elected to Active membership in the Medical Society of the District of Columbia in 1936. He was a diplomate of the American Board of Surgery and a member of the American Medical Association, the Southern Society of Clinical Surgeons, the Society of Medical Consultants to the U.S. Armed Forces, the Washington Academy of Surgery, and the Clinico-Pathological Society. He belonged to the Chevy Chase Country Club.

Surviving him are his wife, Mrs. Sarah Jenkins Briggs, of the home, 2613 Dumbarton Avenue, N.W., and 3 children, all students: Charles, at George Washington University School of Medicine; Christopher, at Trinity College, Hartford, Conn.; and Sally, at Washington University, St. Louis, Mo. He also leaves a sister, Mrs. William Wade, of Denver, Colo., and a brother, A. Sidney Briggs, of Jamestown, Va.

Harry Marshall Fletcher, M.D.

(1906–1968)

Dr. Harry M. Fletcher, ophthalmologist, died, October 14, 1968 at Fairfax Hospital. He had moved to Fairfax, Va., the week preceding his death, where he lived at 1245 4th Street, S.W.

Dr. Fletcher was a native Washingtonian, born January 4, 1906. He received both his Bachelor of Science and Doctor of Medicine degrees from Georgetown University, the latter in 1932. He interned at Georgetown University Hospital.

Following his internship he practiced general medicine in Washington. In 1939 he went abroad to study obstetrics and gynecology at the University of Budapest and the University of Pécs, Hungary.

During World War II he served as a Captain in the Medical Corps of the U.S. Air Force, after which he returned to general practice in Washington.

During 1949 and early 1950 he was abroad again for postgraduate study, this time in ophthalmology at the University of London Postgraduate School of Medicine and at several eye hospitals in London. He returned to Washington to engage in that specialty. Most recently he had offices at 919 18th Street, N.W.

Dr. Fletcher was elected to Active membership in the Medical Society of the District of Columbia in 1935.

He is survived by his mother, Mrs. William Heath, of 4426 Grant Road, N.W., and a brother, Edward W. Fletcher, of Norbeck, Md.

Alexander Contee Thompson, M.D.

(1886–1968)

Dr. A. Contee Thompson, otolaryngologist, died, October 19, 1968 in Doctors Hospital. Since he retired 15 years ago he had divided his time between Babson, Fla., and his home at 104 Russell Avenue, Gaithersburg, Md.

Dr. Thompson, a direct descendant of former Maryland Governor Robert Bowie, was born in Gaithersburg, August 3, 1886. He graduated from St. John's College, Annapolis, Md., in 1907 and from Georgetown University School of Medicine in 1911. He interned at Garfield Hospital for a year.

During World War I he served as a medical officer in the U. S. Navy and continued on duty until 1921.

He practiced in Ashville, N. C., from 1918 until 1933, when he came back to the Washington area. He was on the hospital staff of Episcopal Eye, Ear and Throat Hospital, as junior surgeon from 1934 to 1940 and as associate surgeon from 1940 until he retired from practice.

He was an Active member of the Medical Society of the District of Columbia from 1936 to 1958.

Besides his wife, Mrs. Margaret Hesterberg Thompson, he leaves a daughter, Mrs. J. Grove Seely, of Washington.

Greenville Burton Queen, M.D.

(1908–1968)

Dr. Greenville B. Queen, of Silver Spring, Maryland, an Associate member of the Medical Society of the District of Columbia, died, September 7, 1968 at the Washington Hospital Center.

Dr. Queen was born in Louisa, Kentucky, December 4, 1908. He received his baccalaureate degree from Stanford University in 1929 and his medical degree from Rush Medical College of the University of Chicago in 1933.

Dr. Queen was health officer for Takoma Park, Md., from 1936 to 1940. In addition to Takoma Park, he had practiced in the District of Columbia and Silver Spring, Md., where his offices were last located at 344 University Boulevard, West.

A World War II veteran, he served in the Naval Medical Corps with the rank of Lieutenant Commander and took part in the original landing in Sicily.

He was an Active member of the Montgomery County (Maryland) Medical Society, an Associate member of the D. C. Medical Society since 1946, and a member of the American Academy of General Practice. He was formerly President of the Springbrook Citizens Association in Silver Spring.

Besides his wife, Mrs. Helen Queen, of 110 Warrenton Drive, Silver Spring, he leaves a son, Lt. Greenville Queen, Jr., with the Marines at Cherry Point, N. C., and his mother, Mrs. May Queen, of Louisa, Ky.

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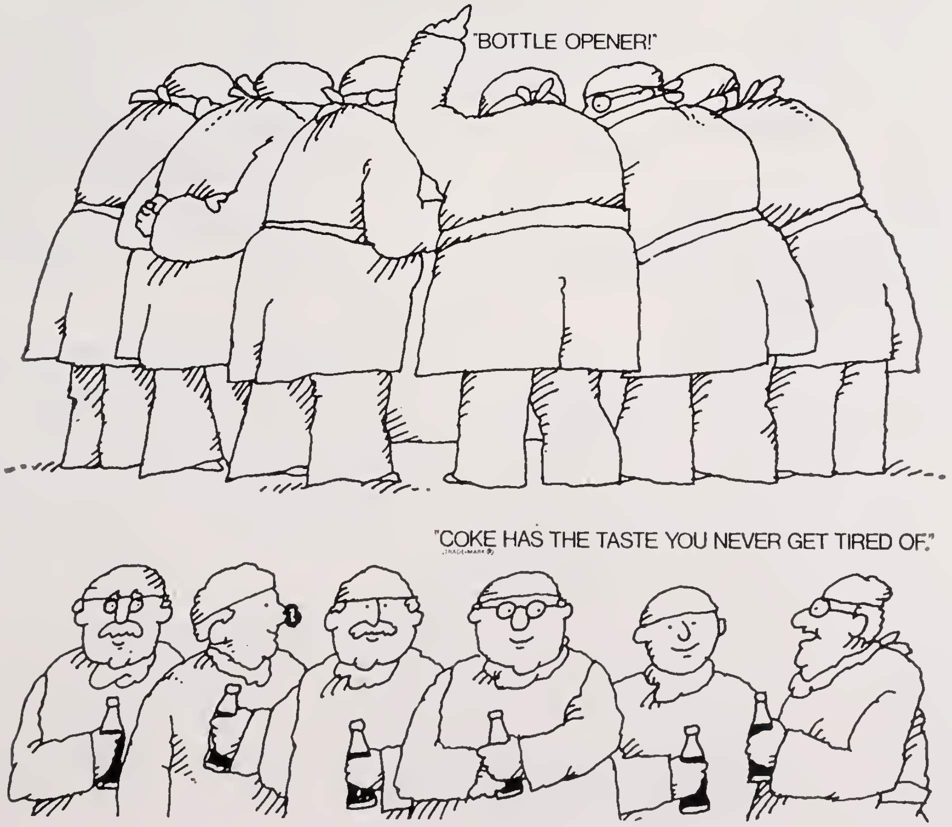
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January 18–23: American Academy of Orthopaedic Surgeons. Americana Hotel, New York. Address: Mr. John K. Hart, Executive Secretary, 29 East Madison Avenue, Chicago, Ill. 60602.

January 23–25: Southern Society for Clinical Investigation. Jung Hotel, New Orleans. Address: Mr. James E. Bryan, 2011 Eye Street, N.W., Washington, D. C. 20006.

February 8–13: International Academy of Proctology (Annual Congress and Teaching Seminar). Hollywood Beach Hotel, Hollywood, Fla. Address: Dr. Alfred J. Cantor, Executive Secretary, 147–41 Sanford Avenue, Flushing, N.Y. 11355.

February 9–10: Congress on Medical Education (65th Annual). Palmer House, Chicago. Address: Dr. C. H. William Ruhe, Secretary, AMA Council on Medical Education, 535 North Dearborn Street, Chicago, Ill. 60610.

February 18–22: American College of Radiology. Regency-Hyatt House Hotel, Atlanta, Ga. Address: Mr. William C. Stronach, Executive Director, 20 North Wacker Drive, Chicago, Ill. 60606.

February 26–March 2: American College of Cardiology. New York Hilton, New York City. Address: Mr. William D. Nelligan, Executive Director, 9650 Rockville Pike, Bethesda, Md. 20014

March 15–19: American Academy of Allergy. Americana Hotel, Bal Harbour, Fla. Address: Mr. James O. Kelly, Executive Secretary, 756 North Milwaukee Street, Milwaukee, Wis. 53202.

March 27–29: Southern Society of Anesthesiologists. Marriott Motor Hotel, Atlanta, Ga. Address: Dr. Richard G. Zepernick, Mercy Hospital, New Orleans, La. 70119.

March 28–29: Third National Congress on the Socio-Economics of Health Care, sponsored by the AMA Council on Medical Service and Division of Health Service. Palmer House, Chicago. Address: Mr. J. E. Garland, Secretary, AMA Council on Medical Service, 535 North Dearborn Street, Chicago, Ill. 60610.

April 20–25: American College of Physicians (50th Annual Session). Conrad Hilton Hotel, Chicago. Address: Dr. Edward C. Rosenow, Jr., Executive Director, American College of Physicians, 4200 Pine Street, Philadelphia, Pa. 19104.

April 28–May 1: American College of Obstetricians and Gynecologists. Americana Hotel, Bal Harbour, Fla. Address: Dr. Michael Newton, Director, 79 West Monroe Street, Chicago, Ill. 60603.

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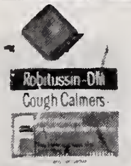
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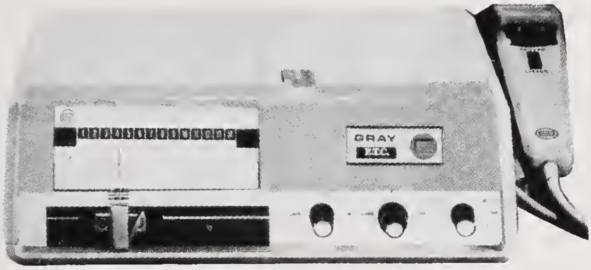

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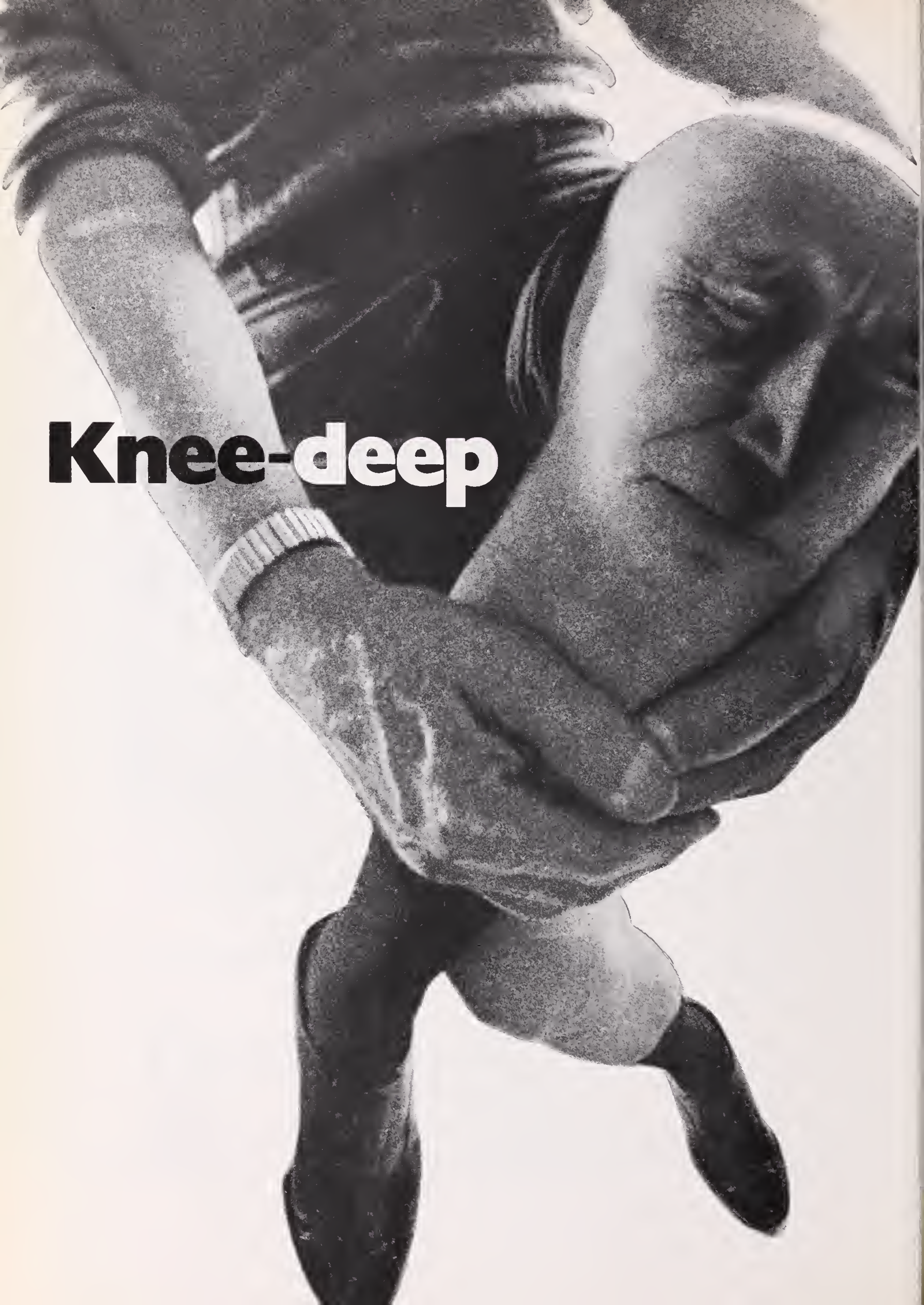
T-text

Knee-deep



TA-6006

in osteoarthritic pain

If aspirin doesn't help, move in with Tandearil.

The trial period is brief: 1 week. Try one tablet q.i.d. at first. Tandearil usually starts working within 3 to 4 days. When response occurs, as little as 1 or 2 tablets daily may hold back pain and stiffness, and increase joint motion.

On the next page is a summary of adverse reactions, contraindications, warning and precautions.

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It can help get his mind off his knee.

Please review full Prescribing Information carefully before prescribing.

For osteoarthritic knees, spines, shoulders, hips, etc.:

Tandearil®
oxyphenbutazone

Tandearil, oxyphenbutazone:
For brief summary see next page.

Geigy

TA-6006

Tandearil®
oxyphenbutazone

Contraindications: Edema; danger of cardiac decompensation; history or symptoms of peptic ulcer; renal, hepatic or cardiac damage; history of drug allergy; history of blood dyscrasia. The drug should not be given when the patient is senile or when other potent drugs are given concurrently.

Warning: This drug is an analog of phenylbutazone; sensitive patients may be cross-reactive. If coumarin-type anticoagulants are given simultaneously, watch for excessive increase in prothrombin time. Instances of severe bleeding have occurred. Persistent or severe dyspepsia may indicate peptic ulcer; perform upper gastrointestinal x-ray diagnostic tests if drug is continued. Pyrazole compounds may potentiate the pharmacologic action of sulfonylurea, sulfonamide-type agents and insulin. Carefully observe patients receiving such therapy. Use with caution in the first trimester of pregnancy, and in patients with thyroid disease.

Precautions: Before prescribing, carefully select patients, avoiding those responsive to routine measures as well as contraindicated patients. Obtain a detailed history and a complete physical and laboratory examination, including a blood count. The patients should not exceed recommended dosage, should be closely supervised and should be warned to discontinue the drug and report immediately if fever, sore throat, or mouth lesions (symptoms of blood dyscrasia), sudden weight gain (water retention), skin reactions, black or tarry stools or other evidence of intestinal hemorrhage occur. Make complete blood counts at weekly intervals during early therapy and at 2-week intervals thereafter. Discontinue the drug immediately and institute countermeasures if the white count changes significantly, granulocytes decrease, or immature forms appear. Use greater care in the elderly and in hypertensives.

Adverse Reactions: The more common are nausea and edema. Swelling of the ankles or face may be minimized by withholding dietary salt, reduction in dosage or use of diuretics. In elderly patients and in those with hypertension, the drug should be discontinued with the appearance of edema. The drug has been associated with peptic ulcer and may reactivate a latent peptic ulcer. The patient should be instructed to take doses immediately after meals or with milk to minimize gastric upset. Drug rash occasionally occurs. If it does, promptly discontinue the drug. Agranulocytosis, exfoliative dermatitis, Stevens-Johnson syndrome, Lyell's syndrome (toxic necrotizing epidermolysis), or a generalized allergic reaction similar to a serum sickness syndrome may occur and require permanent withdrawal of medication. Agranulocytosis can occur suddenly in spite of regular, repeated normal white counts. Stomatitis, salivary gland enlargement, vomiting, vertigo and languor may occur. Leukemia and leukemoid reactions have been reported. While not definitely attributable to the drug, a causal relationship cannot be excluded. Thrombocytopenic purpura and aplastic anemia may occur. Confusional states, agitation, headache, blurred vision, optic neuritis and transient hearing loss have been reported, as have hyperglycemia, hepatitis, jaundice, hypersensitivity angiitis, pericarditis and several cases of anuria and hematuria. With long-term use, reversible thyroid hyperplasia may occur infrequently. Moderate lowering of the red cell count due to hemodilution may occur.

Dosage in Osteoarthritis: Initial: 3 to 6 tablets daily in divided doses. Usually unnecessary to exceed 4 tablets daily. A trial period of one week is considered adequate to determine the therapeutic effect of the drug. Maintenance: Effective level often achieved with 1 or 2 tablets daily, should not exceed 4 tablets daily.

In selecting appropriate dosage in any specific case, consideration should be given to the patient's weight, general health, age and any other factors influencing drug response.

Availability: Tan, round, sugar-coated tablets of 100 mg. in bottles of 100 and 1000.
(B)R-46-800-A

For complete details, please see full Prescribing Information.

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Dilantin®
(diphenylhydantoin)
PARKE-DAVIS

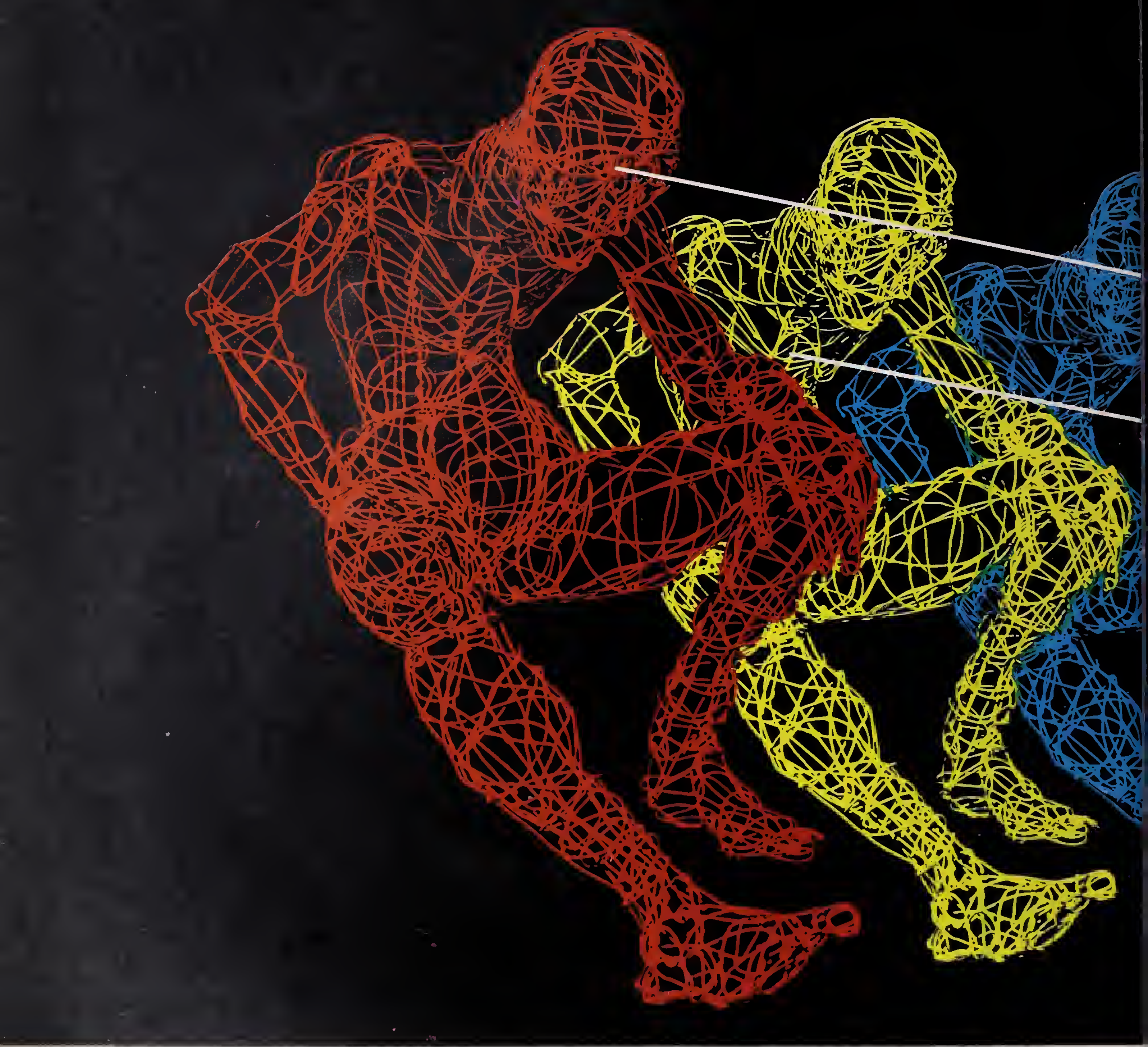
In untold thousands of epileptic patients... Dilantin has been, and continues to be, the bedrock of therapy.

DILANTIN is useful in the treatment of grand mal epilepsy and certain other convulsive states. Its use will prevent or greatly reduce the incidence and severity of convulsive seizures in a substantial percentage of epileptic patients, without the hypnotic and narcotizing effects of many anticonvulsant drugs.
PRECAUTIONS: Periodic examination of the blood is advisable. Nystagmus in combination with diplopia and ataxia indicates dosage should be reduced. The possibility of toxic effects during pregnancy has not been explored. ADVERSE REACTIONS: Allergic phenomena such as polyarthropathy, fever, skin eruptions, and acute generalized morbilliform eruptions with or without fever. Rarely, dermatitis goes on to exfoliation with hepatitis, and further dosage is contraindicated. Gingival hypertrophy, hirsutism, and excessive motor activity are occasionally encountered. During initial treatment, side effects may include gastric distress, nausea, weight loss, nervousness, sleeplessness, feeling of unsteadiness. Macrocytosis, megaloblastic anemia, leukopenia, granulocytopenia, thrombocytopenia, pancytopenia, agranulocytosis, and aplastic anemia have been reported. Nystagmus, lymphadenopathy, lupus erythematosus, erythema multiforme (Stevens-Johnson syndrome), and a syndrome resembling infectious mononucleosis with jaundice have occurred.
DILANTIN is supplied in several forms including Kapseals® containing 0.1 Gm. and 0.03 Gm. diphenylhydantoin sodium.
Parke, Davis & Company, Detroit, Michigan 48232

The color combinations of the banded capsules are Parke-Davis trademarks. The orange-banded white capsule identifies Parke-Davis 0.1 Gm. diphenylhydantoin sodium; the pink-banded white capsule 0.03 Gm. diphenylhydantoin sodium.

PARKE-DAVIS 015R67

a comprehensive hematinic



escription: Each Pulvule® contains—

pecial Liver-Stomach Concentrate, Lilly (containing Intrinsic Factor)	150	mg.
obalamin Concentrate, N.F., equivalent to Cobalamin (The total vitamin B_{12} activity in the Special Liver-Stomach Concentrate, Lilly, and the Cobalamin Concentrate, N.F., is 15 micrograms.)	7.5	mcg.
on, Elemental (as Ferrous Fumarate)	110	mg.
scorbic Acid (Vitamin C)	75	mg.
olic Acid	1	mg.

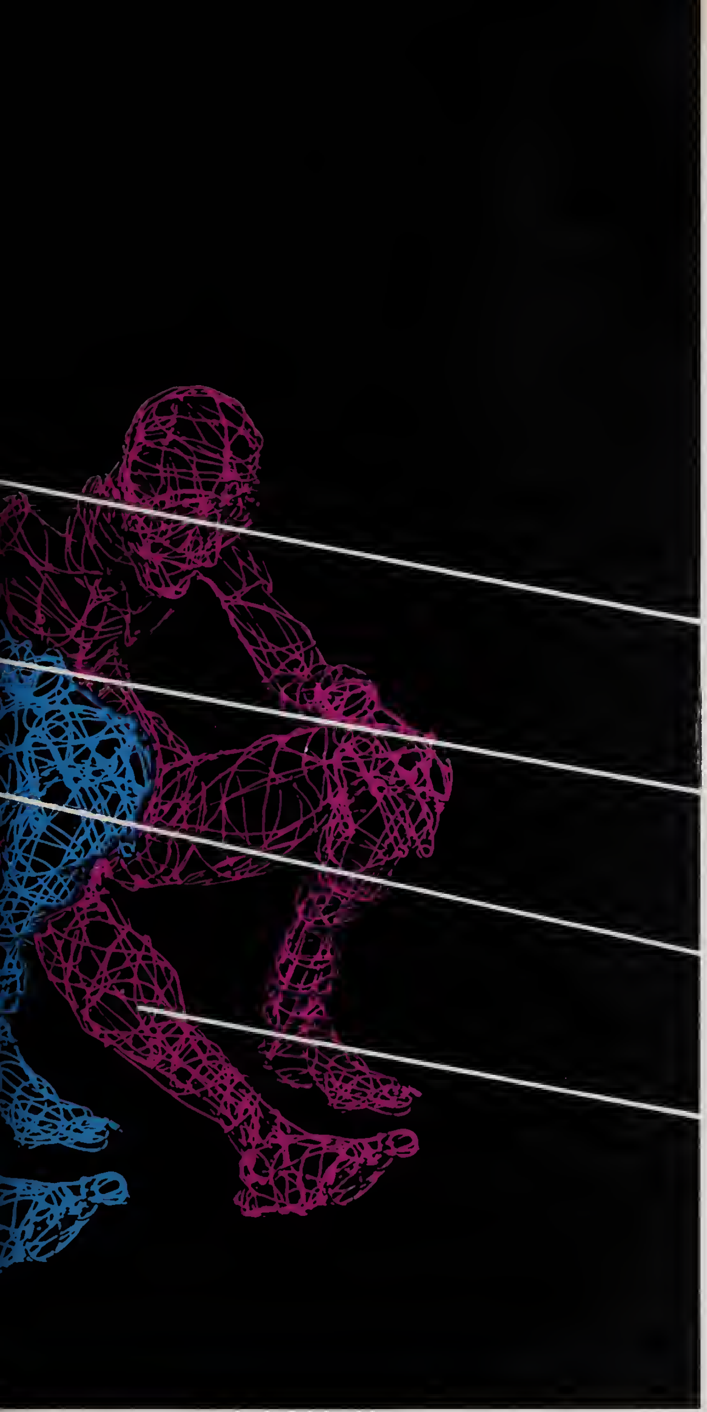
ndications: Trinsicon® (hematinic concentrate with intrinsic factor, Lilly) is a multifactor preparation effective in the treatment f anemias that respond to oral hematinics, including pernicious nemia and other megaloblastic anemias and also iron-deficiency nemia.

Contraindications: Hemochromatosis and hemosiderosis.

Precautions: Anemia is a manifestation that requires appropriate investigation to determine its cause or causes.

In pernicious anemia, the use of folic acid without adequate vitamin B_{12} therapy may result in hematologic remission but neurological progression. Adequate doses of vitamin B_{12} (parenteral, or oral with potent intrinsic factor as in Trinsicon® [hematinic concentrate with intrinsic factor, Lilly]) usually prevent, halt, or improve the neurological changes.

As with all preparations containing intrinsic factor, resistance may develop in some cases of pernicious anemia to the potentiation of absorption of physiological doses of vitamin B_{12}. If resistance occurs, parenteral therapy, or oral therapy with so-called massive doses of vitamin B_{12}, may be necessary. No single regimen fits all cases, and the status of the patient observed in follow-up is the final criterion for adequacy of therapy. Periodic



You can treat combined deficiencies with

Trinsicon®

—the multifactor hematinic

Vitamin B_{12} plus intrinsic factor (15 mcg. B_{12} activity)—helps provide adequate levels of this important vitamin.

Folic acid (1 mg.)—treats nutritional macrocytic anemias and/or malabsorption syndromes.

Ascorbic acid (75 mg.)—augments the conversion of folic acid to its active form and helps iron absorption.

Iron (110 mg.)—treats hypochromic anemia.

clinical and laboratory studies are considered essential and are recommended.

Adverse Reactions: In rare instances, iron in therapeutic doses produces gastro-intestinal reactions, such as diarrhea or constipation. Reducing the dose and administering it with meals will minimize these effects.

In extremely rare instances, skin rash suggesting allergy has followed oral administration of liver-stomach material. Instances of apparent allergic sensitization have also been reported after oral administration of folic acid.

Dosage: One Pulvule twice a day. (Two Pulvules daily produce a standard response in the average uncomplicated case of pernicious anemia.)

How Supplied: Pulvules Trinsicon® (hematinic concentrate with intrinsic factor, Lilly), in bottles of 60 and 500. [032568]

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Lilly

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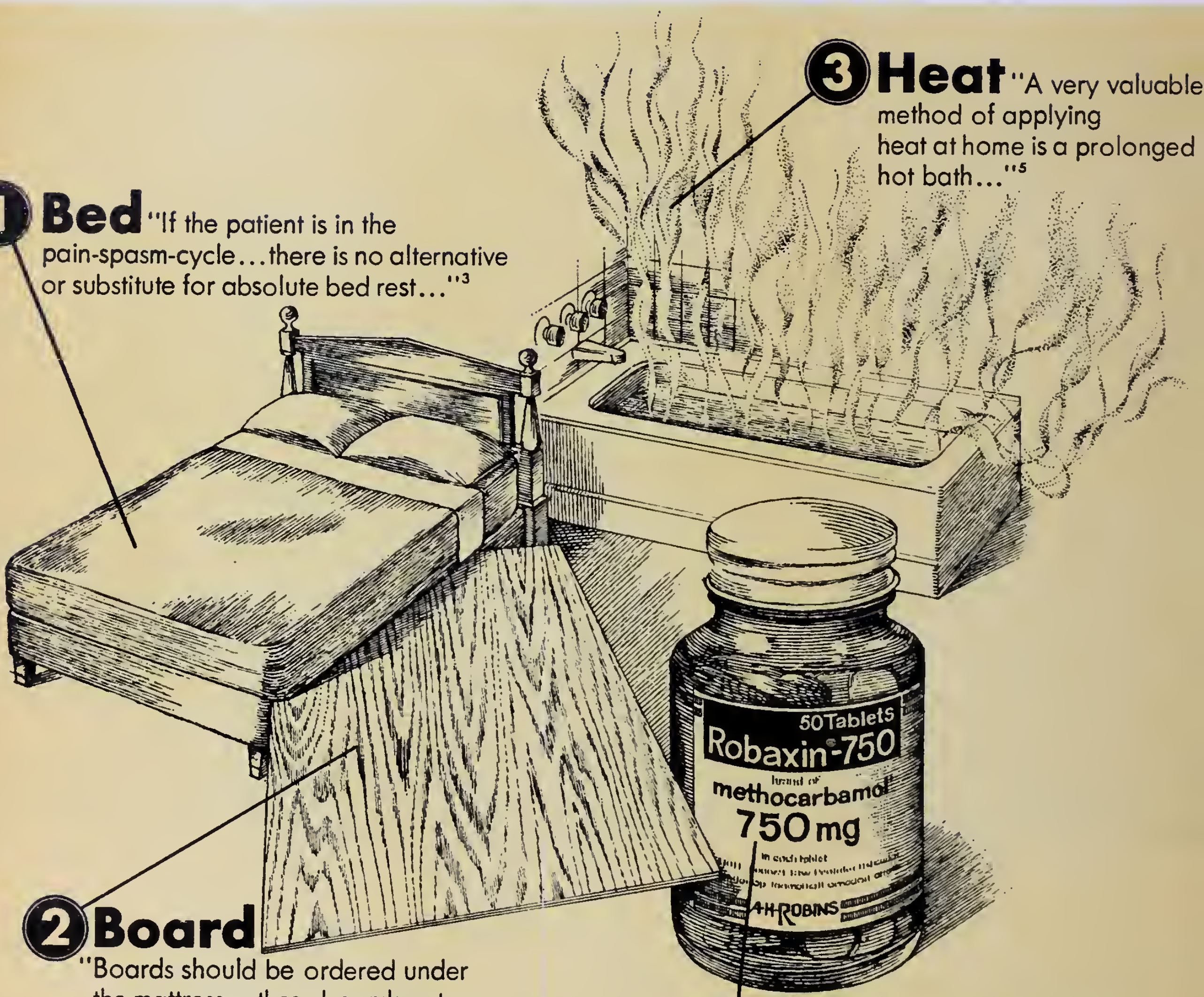

① Bed "If the patient is in the pain-spasm-cycle...there is no alternative or substitute for absolute bed rest..."[3]

② Board "Boards should be ordered under the mattress...these boards act by immobilizing the spine..."[4]

③ Heat "A very valuable method of applying heat at home is a prolonged hot bath..."[5]

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(methocarbamol, 750 mg. capsule-shaped tablets) A well-tolerated[6] skeletal muscle relaxant, methocarbamol helps relieve spasm "...without interfering with normal tone and movement."[7] And there is little likelihood of sedation.[6]

Indicated for relief of skeletal muscle spasm. **Contraindicated** in hypersensitive patients. **Side Effects** (lightheadedness, dizziness, drawsiness, nausea) may occur rarely, but usually disappear on reduced dosage. Hypersensitivity reactions develop infrequently. See product literature for further details. **Also available:** Robaxin® Tablets (methocarbamol, 500 mg.) Robaxin Injectable (methocarbamol, 1 Gm./10 cc.)
References: (1). Godfrey, C.M.: Applied Therap. 8:950, 1966. (2). Gottschalk, L.A.: GP 33:91, 1966. (3). Rowe, M.L.: J. Occup. Med. 2:219, 1960. (4). Cozen, L.: South Dakota J. Med. 18:26, 1965. (5). Sota-Hall, R.: Med. Sc. 14:23, 1963. (6). Weiss, M. and Weiss, S.: J. Am. Osteopath. A. 62:142, 1962. (7). Feuer, S.G., et *al.*: New York J. Med. 62:1985, 1962.

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ready for bed...
but not for sleep

...because psychic tension may not stop at night

The calming action of Valium (diazepam) helps counteract psychic tension and reduce overreaction to stresses during the day. Often the t.i.d. dosage schedule is enough to prevent build-up of tenseness that may interfere with sleep at night.

However, when psychic tension does contribute to sleeplessness, Valium can be especially useful. A tablet at bedtime, added to the daytime t.i.d. dosage, can help your patient be ready for bed *and* for sleep.

Before prescribing, please consult complete product information, a summary of which follows:

Indications: Tension and anxiety states; somatic complaints which are concomitants of emotional factors; psychoneurotic states manifested by tension, anxiety, apprehension, fatigue, depressive symptoms or agitation; acute agitation, tremor, delirium tremens and hallucinosis due to acute alcohol withdrawal; adjunctively in skeletal muscle spasm due to reflex spasm to local pathology, spasticity caused by upper motor neuron disorders, athetosis, stiff-man syndrome, convulsive disorders (not for sole therapy).

Contraindicated: Known hypersensitivity to the drug. Children under 6 months of age. Acute narrow angle glaucoma.

Warnings: Not of value in psychotic patients. Caution against hazardous occupations requiring complete mental alertness. When used adjunctively in convulsive disorders, possibility of increase in frequency and/or severity of grand mal seizures may require increased dosage of standard anticonvulsant medication; abrupt withdrawal may be associated with temporary increase in frequency and/or severity of seizures. Advise against simultaneous ingestion of alcohol and other CNS depressants. Withdrawal symptoms have occurred following abrupt discontinuance. Keep addiction-prone individuals under careful surveillance because of their predisposition to habituation and dependence. In pregnancy, lactation or women of childbearing age, weigh potential benefit against possible hazard.

Precautions: If combined with other psychotropics or anticonvulsants, consider carefully pharmacology of agents employed. Usual precautions indicated in patients severely depressed, or with latent depression, or with suicidal tendencies. Observe usual precautions in impaired renal or hepatic function. Limit dosage to smallest effective amount in elderly and debilitated to preclude ataxia or oversedation.

Side Effects: Drowsiness, confusion, diplopia, hypotension, changes in libido, nausea, fatigue, depression, dysarthria, jaundice, skin rash, ataxia, constipation, headache, incontinence, changes in salivation, slurred speech, tremor, vertigo, urinary retention, blurred vision. Paradoxical reactions such as acute hyperexcited states, anxiety, hallucinations, increased muscle spasticity, insomnia, rage, sleep disturbances, stimulation, have been reported; should these occur, discontinue drug. Isolated reports of neutropenia, jaundice; periodic blood counts and liver function tests advisable during long-term therapy.

Valium®
(diazepam)

ROCHE
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LABORATORIES
Division of Hoffmann-La Roche Inc.
Nutley, New Jersey 07110

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